The spectrum of involuntary vocalizations in humans

Mainka, Tina; Balint, Bettina; Goevert, Felix; Kurvits, Lille; van Riesen, Christoph; Kuehn, Andrea A.; Tijssen, Marina A. J.; Lees, Andrew J.; Mueller-Vahl, Kirsten; Bhatia, Kailash P.

Published in:
Movement Disorders

DOI:
10.1002/mds.27855

REVIEW

The Spectrum of Involuntary Vocalizations in Humans: A Video Atlas

Tina Mainka, MD,[1] Bettina Balint, MD,[2,3] Felix Gövert, MD,MSc,[4] Lille Kurvits, MD,[1] Christoph van Riesen, MD,[1,5] Andrea A. Kühn, MD, PhD,[1] Marina A.J. Tijssen, MD, PhD,[6] Andrew J. Lees, FRCP, F.Med.Sci.,[7] Kirsten Müller-Vahl, MD,[8] Kailash P. Bhatia, MD, FRCP,[2] and Christos Ganos, MD[1*]

[1]*Department of Neurology, Charité University Medicine Berlin, Berlin, Germany*
[2]*Department of Clinical and Movement Neurosciences, Queen Square Institute of Neurology, University College London, London, UK*
[3]*Department of Neurology, University Hospital Heidelberg, Heidelberg, Germany*
[4]*Department of Neurology, University Hospital Schleswig-Holstein, Christian-Albrechts-University, Kiel, Germany*
[5]*Department of Neurology, University Medicine Göttingen, Göttingen, Germany*
[6]*Department of Neurology, University Medical Centre Groningen, University of Groningen, Groningen, The Netherlands*
[7]*Reta Lila Weston Institute of Neurological Studies, UCL, Institute of Neurology, London, UK*
[8]*Clinic of Psychiatry, Socialpsychiatry and Psychotherapy, Hannover Medical School, Hannover, Germany*

ABSTRACT: In clinical practice, involuntary vocalizing behaviors are typically associated with Tourette syndrome and other tic disorders. However, they may also be encountered throughout the entire tenor of neuropsychiatry, movement disorders, and neurodevelopmental syndromes. Importantly, involuntary vocalizing behaviors may often constitute a predominant clinical sign, and, therefore, their early recognition and appropriate classification are necessary to guide diagnosis and treatment. Clinical literature and video-documented cases on the topic are surprisingly scarce. Here, we pooled data from 5 expert centers of movement disorders, with instructive video material to cover the entire range of involuntary vocalizations in humans. Medical literature was also reviewed to document the range of possible etiologies associated with the different types of vocalizing behaviors and to explore treatment options. We propose a phenomenological classification of involuntary vocalizations within different categorical domains, including (1) tics and tic-like vocalizations, (2) vocalizations as part of stereotypies, (3) vocalizations as part of dystonia or chorea, (4) continuous vocalizing behaviors such as groaning or grunting, (5) pathological laughter and crying, (6) vocalizations resembling physiological reflexes, and (7) other vocalizations, for example, those associated with exaggerated startle responses, as part of epilepsy and sleep-related phenomena. We provide comprehensive lists of their associated etiologies, including neurodevelopmental, neurodegenerative, neuroimmunological, and structural causes and clinical clues. We then expand on the pathophysiology of the different vocalizing behaviors and comment on available treatment options. Finally, we present an algorithmic approach that covers the wide range of involuntary vocalizations in humans, with the ultimate goal of improving diagnostic accuracy and guiding appropriate treatment.

Key Words: involuntary vocalizations; movement disorders; vocalizing behavior

Correction added on October 31, 2019, after first online publication: Article text edits were made in the article for style purposes.

***Correspondence to:** Christos Ganos, MD, Movement Disorders and Body Control Lab, Movement Disorders and Neuromodulation Unit, Department of Neurology, Charité University Medicine Berlin, Charitéplatz 1, 10117 Berlin, Germany; Email: christos.ganos@charite.de

Relevant conflicts of interest/financial disclosures: The authors disclose no conflicts of interest regarding this article.

Funding agencies: This research project was supported by a grant from the VolkswagenStiftung (Freigeist) held by C.G.

Received: 4 June 2019; **Revised:** 22 July 2019; **Accepted:** 21 August 2019

Published online 25 October 2019 in Wiley Online Library (wileyonlinelibrary.com). DOI: 10.1002/mds.27855

The ability to vocalize has only been a fairly recent evolutionary acquisition and was a prerequisite for the development of verbal communication in our species.[1] Our acquired repertoire of vocalizations ranges from simple sounds related to physiological reflexes (eg, sneezing) and emotional responses (eg, crying, laughing) to the intended articulation of words that are meant to express specific communicative content.[2] In all these instances, vocalizations are typically context specific and adaptive to environmental stimuli. However, the occurrence of vocalizing behaviors in the absence of these qualities typically signifies pathology and most often constitutes a major cause of distress.

Medical literature and clinical practice have historically associated abnormal vocalizing behaviors with tic disorders, as for example, Tourette syndrome (TS), of which they are also an essential part of the diagnostic criteria.[3] However, involuntary vocalizations may also be encountered throughout the entire tenor of neuropsychiatric disorders, to include movement disorders, neurodegenerative and neurodevelopmental syndromes, and functional neurological disorders. Ictal phenomena in epileptic disorders may also present with vocalizing behaviors. Although in many of these disorders, abnormal vocalizations will often be only one feature of a range of abnormal motor behaviors and clinical signs, in some cases, they may constitute the sole clinical finding. Here, their early recognition and appropriate classification are paramount for guiding diagnostic reasoning and informing therapeutic decisions. However, beyond tic disorders and TS, the clinical literature on the topic remains sparse,[2] and video-documented cases are particularly rare.

Over a period of several years, we came across a number of patients in whom abnormal vocalizations were the predominant reason for clinical presentation. Given the difficulties in the phenomenological classification of vocalizing behaviors, we here provide a clinical overview of the range of involuntary vocalizations in humans, together with 29 informative video-documented cases, to illustrate both typical and more unusual clinical examples. Our goal is to inform our colleagues from the neighboring fields of neurology, neuropsychiatry, and psychiatry on the phenomenological spectrum and diagnostic conditions associated with involuntary vocalizations, discuss their pathophysiology, and provide treatment recommendations where possible.

Methods

Data from 5 expert centers of movement disorders across Europe (Department of Neurology, Charité University Medicine Berlin, Berlin, Germany; Department of Clinical and Movement Neurosciences, Queen Square Institute of Neurology, University College London, London, UK; Department of Neurology, University Hospital Schleswig-Holstein, Christian-Albrechts-University, Kiel, Germany; Department of Neurology, University Medical Centre Groningen, University of Groningen, Groningen, The Netherlands; Clinic of Psychiatry, Socialpsychiatry and Psychotherapy, Hannover Medical School, Hannover, Germany) were pooled for this study. Cases of patients in whom involuntary vocalizations predominated in clinical presentation and for whom video material was available were first collected and reviewed. We selected the cases that exemplified distinct phenomenological characteristics of different vocalizing behaviors. We also reviewed the literature to identify the range of possible etiologies associated with the different types of vocalizing behaviors that we included and to explore treatment options. Based on our clinical experience and the available material we gathered, we also provide practical treatment recommendations where possible. Signed patient consent was obtained for videos of all patients that we present here.

Tics and Tic-Like Vocalizations

Tics are defined as movements or sounds that resemble physiological motor behaviors, but are typically inopportune to social context and appear sudden, repetitive, and often exaggerated.[4] Tic vocalizations — commonly termed vocal or phonic tics — may include any possible sound (eg, sniffing, coughing, throat clearing, whistling, or grunting), word, or sentence and are most commonly encountered within the spectrum of primary tic disorders, as TS (Video 1A–C). In these patients, tics, including phonic and vocal behaviors, are typically preceded by premonitory urges and can be suppressed voluntarily.[4-7] Individuals with autism spectrum disorders (ASD) may also present with vocal tics, and indeed an overlap between primary tic disorders and autistic features has been reported in the medical literature.[8,9] However, in ASD premonitory urges and overall vocal tic awareness may be reduced compared to people with primary tic disorders and TS.[10] Klinefelter,[11] fragile X,[12] and Adams-Oliver syndrome,[13] as well as monosomy 9p[14] and trisomy 16p[15] are documented genetic causes of other neurodevelopmental disorders that may manifest phonic/vocal tics. Neurodegenerative syndromes may also present with phonic or vocal tics, for example, in Huntington's disease (Video 1D). Here, vocalizing behaviors such as grunting tics are often characteristic (Video 1E,F),[16-18] and although the distinction of tics from choreic sounds (also see the section on Vocalizations as Part of Dystonia, Chorea, and Other Dyskinesias) may often be difficult, some patients describe the presence of premonitory urges preceding vocal tics (case example of video 1E). Furthermore, vocal tics have been documented in patients with chorea-acanthocytosis because of VPS13A mutations[19-21] (Video 1G), Amyotrophic lateral sclerosis (ALS) frontotemporal dementia (FTD) overlap syndromes,[22] progressive supranuclear palsy (PSP),[23] and pantothenate kinase-associated neurodegeneration (PKAN).[24] Neurometabolic disorders such as Wilson's disease or phenylketonuria,[25,26] focal brain lesions,[27-34] infectious,[35-37] and other autoimmune diseases[38-41] are additional causes of vocal tics (see Table 1). Finally, phonic or vocal tics may also be drug-induced, either directly related to the acute effects of drugs[42-46] (eg, cocaine[46]) or as a long-term consequence, such as in tardive tic disorders[47-49] (Video 1H).

A final etiological category includes functional neurological disorders. Previous literature on such cases refers to repetitive sounds resembling vocal tics as tic-like vocalizations and offers clinical clues to distinguish the 2 types of behaviors.[50-53] Abrupt symptom onset, typically in

TABLE 1. Spectrum of involuntary vocalizations in humans, their descriptions, and etiologies

Vocalization	Description	Possible etiology
Tics and tic-like vocalizations	Sudden, exaggerated, repetitive, and inopportune to social context sounds (eg, sniffing, coughing, throat clearing, whistling, grunting) or words[b]	— Primary tic disorders (eg, TS) — Other neurodevelopmental disorders (eg, ASD, Klinefelter, fragile X, Adams-Oliver syndrome, monosomy 9p, trisomy 16p) — Neurodegenerative disorders (eg, HD, chorea-acanthocytosis, ALS-FTD overlap syndromes, PSP, PKAN) — Neurometabolic disorders (eg, Wilson's disease, PKU) — Focal brain lesions (eg, after head trauma, arteriovenous hemorrhage, following cardiac surgery, after temporal lobectomy, in osmotic demyelination syndrome, after carbon monoxide poisoning, postinfectious (VZV encephalitis)) — Infectious (eg, HIV, HSV, rubella virus) — Autoimmune (eg, postinfectious, MS, SLE, Behcet's disease, antiphospholipid syndrome) — Drug-induced (eg, carbamazepine, lamotrigine, bupropion, cocaine) — Tardive (eg, antipsychotics) — Functional neurological disorders
• Klazomania	Compulsive shouting episodes	— Primary tic disorders (eg, TS) — Focal brain lesions (eg, carbon monoxide poisoning) — Others (eg, depressive disorders, postencephalitic parkinsonism) — Functional neurological disorders
• Palilalia	Repetition of one's own syllables, words, or phrases 2 or more times in a row	— Primary tic disorders (eg, TS) — Other neurodevelopmental disorders (eg, ASD, trisomy 16p) — Neurodegenerative disorders (eg, Alzheimer's disease, PSP, chorea-acanthocytosis, VCP proteinopathy, PD[a]) — Focal brain lesions (eg, ischemic, hemorrhagic, after stereotaxic thalamotomy, after severe head trauma, after carbon monoxide poisoning, after respiratory acidosis, associated with extensive intracerebral calcifications, postinfectious [VZV encephalitis]) — Ictal — Autoimmune (eg, steroid-responsive encephalopathy) — Drug-induced (eg, clozapine, cefepime) — Others (eg, early-onset schizophrenia, membranous lipodystrophy, postencephalitic parkinsonism) — Functional neurological disorders (including startle syndromes, eg, Latah)
• Echolalia	Imitative repetition of sounds, words, or phrases in the absence of explicit awareness	— Primary tic disorders (eg, TS) — Other neurodevelopmental disorders (eg, ASD, Rubinstein-Taybi, fragile X, Williams syndrome, trisomy 16p) — Neurodegenerative disorders (eg, DLB, FTD, Alzheimer's disease, HD, CJD, PSP-CBS, CBD, familial progressive subcortical gliosis, chorea-acanthocytosis) — Neurometabolic disorders (eg, Wilson's disease, NPC, encephalopathy in D-lactic acidosis, after liver transplantation) — Focal brain lesions (eg, ischemic, after severe head trauma, after carbon monoxide poisoning) — Infectious (eg, cerebral malaria) — Autoimmune (eg, Hashimoto's encephalopathy, MS, NMDA-receptor encephalitis, SLE) — Drug-induced (eg, isoniazid, topiramate, ofloxacin, methoxphenidine, cocaine, designer tryptamine, phencyclidine) — Functional neurological disorders (including startle syndromes, eg, Jumping Frenchmen of Maine, Latah, Ragin' Cajuns of Louisiana) — Others (eg, encephalitis lethargica, catatonia)
• Coprolalia	Unintended utterance of obscenities and socially inappropriate and derogatory remarks	— Primary tic disorders (eg, TS) — Other neurodevelopmental disorders (eg, Kleine-Levin syndrome, fragile X syndrome) — Neurodegenerative disorders (eg, FTD, Alzheimer's disease, choreo-acanthocytosis) — Focal brain lesions (eg, after carbon monoxide poisoning) — Ictal — Functional neurological disorders — Others (eg, encephalitis lethargica)
Vocalizations as part of stereotypies	Vocalizations associated with repetitive, non-goal-directed, and distractible movement patterns	— Physiological, normal development — Neurodevelopmental disorders (eg, ASD, 15q13.3 microdeletion, Rett syndrome) — Others (eg, schizophrenia)

(Continues)

TABLE 1. Continued

Vocalization	Description	Possible etiology
Vocalizations as part of dystonia, chorea, and other dyskinesias	Phonic or vocal phenomena due to hyperkinetic movements, as chorea, dystonia, and other dyskinesias[b]	— Neurodegenerative disorders (eg, HD, chorea-acanthocytosis) — Autoimmune (eg, postinfectious) — Drug-induced (eg, antipsychotics, metoclopramide, lenalidomide)
Continuous vocalizations such as groaning, moaning, grunting, and shrieking	Continuous or repetitive groaning, moaning, grunting, and shrieking in the absence of appropriate context	— Neurodegenerative disorders (eg, Alzheimer's disease, vascular dementia, HD, PD, PSP) — Neurometabolic disorders (eg, acquired hepatocerebral degeneration) — Functional neurological disorders
Pathological laughter and crying	Laughter and crying occurring detached from emotional content	— Primary tic disorders (eg, TS) — Other neurodevelopmental disorders (eg, Angelman syndrome, partial trisomy 16p, Rett-like syndromes) — Neurodegenerative disorders (eg, ALS, FTD, Alzheimer's disease, primary progressive aphasia, MSA-C, CJD, SCA17, HD) — Focal brain lesions (eg, cerebrovascular disease, traumatic brain lesions) — Ictal (eg, gelastic seizures) — Autoimmune (eg, acute disseminated encephalomyelitis, MS) — Drug induced (eg, intravenous sodium valproate)
Vocalizations resembling physiological reflexes	Repetitive sounds such as belching, sniffing, coughing, wheezing[b]	— Physiological (eg, contagious yawning, groaning during sexual intercourse) — Primary tic disorders (eg, TS) — Neurodegenerative disorders (eg, as OFF symptom in PD) — Focal brain lesions (eg, ischemic) — Ictal (eg, seizure-ending signs, temporal lobe seizures) — Infectious (eg, herpes simplex encephalitis) — Functional neurological disorders
Others	Broad range of involuntary vocalizations not clearly belonging in any of the previous categories[b]	— Culture-bound startle syndromes — Functional neurological disorders (eg, exaggerated stimulus-triggered responses) — Ictal (eg, ictal cry, animal noises, singing, and humming) — Sleep related (eg, snoring, catathrenia, stridor [eg, in MSA, anti-IgLON5 disease, SCA17]), — Night terrors — Sleep-related hypermotor seizures — REM sleep disorder in primary tic disorders (eg, TS), neurodevelopmental disorders (eg, ASD), neurodegenerative disorders (eg, PD, MSA, DLB, FTD, ALS, SCA3, xeroderma pigmentosum, HD), focal brain lesions (eg, brain stem ischemia, tumors) autoimmune disorders (eg, MS, Guillain-Barré syndrome, paraneoplastic), and others (eg, narcolepsy, epilepsy, posttraumatic stress disorder)

ALS, amyotrophic lateral sclerosis; ASD, autism spectrum disorder; CBD, corticobasal degeneration; CBS, corticobasal syndrome; CJD, Creutzfeld-Jacob disease; DLB, dementia with Lewy bodies; FTD, frontotemporal dementia; HD, Huntington's disease; HIV, human immunodeficiency virus; HSV, herpes simplex virus; MS, multiple sclerosis; MSA-C, multiple system atrophy–cerebellar type; NPC, Niemann Pick type C; PD, Parkinson's disease; PKAN, pantothenate kinase-associated neurodegeneration; PKU, phenylketonuria; PSP, progressive supranuclear palsy; REM, rapid eye movement; SCA, spinocerebellar ataxia; SLE, systemic lupus erythematosus; TS, Tourette syndrome; VCP, valosin-containing-protein; VZV, varicella zoster virus.
[a]May occur in association with peak-dose levodopa or as side effect of deep brain stimulation.
[b]Some sounds are mediated by supraglottic structures without involvement of the larynx.

adulthood, absence of premonitory urges, lack of suppressibility, and atypical response to anti-tic medication, alongside the presence of further functional movement disorders and medically unexplained symptoms, are indeed characteristic red flags that should prompt the consideration of a functional etiology. However, even with these helpful aids, correct diagnostic labeling and etiological distinction may often be challenging, particularly in cases in which both tics and tic-like movements or sounds may co-occur.[4,54]

Klazomania

The term *klazomania* (after the Greek word for "crying") was first coined in 1925 by Benedek, a German psychiatrist, who described a patient with postencephalitic parkinsonism and involuntary attacks of compulsive paroxysmal shouting.[55] The shouting behaviors were described as extremely loud and not related to the ongoing mental state of the patient. They occurred in bouts and could last for several hours. Syllables, vowels, single words, and sometimes noises, described in the original report as "carnivorous" animal sounds, were noted.[55] Palilalic behaviors (see below) were also described in this patient, who was able to briefly suppress the involuntary vocalizations with forceful breathing. Inappropriate shouting is also a well-documented feature in TS[56] (Video 1I) and functional neurological disorders (Video 1J). Other associations of klazomania include depression[57-59] and carbon

monoxide poisoning.[60,61] Clearly, as in the distinction of tics from tic-like vocalizations, beyond phenomenological observation of vocalizing behaviors, historical information and the presence of additional clinical features are crucial to distinguish between the different etiological categories.

Palilalia

Palilalia is the involuntary repetition of one's own phrases, words, or syllables 2 or more times in a row.[62] Typically, palilalic utterances decrease in volume with the increasing number of repetitions.[63] Sometimes, the repetitions are also uttered with an accelerating speed.[62,64]

In 1908, Souques first described palilalia in a patient with an ischemic stroke of the right hemisphere.[62] Since then, palilalia, which is typically documented in up to a third of patients with TS[56,65-67] (Video 1A), was reported in patients with other neurodevelopmental disorders such as ASD[68] or trisomy 16p[15] and neurodegenerative disorders, such as PSP,[69,70] dementia of the Alzheimer's type,[71] valosin-containing-protein proteinopathy,[72] or chorea-acanthocytosis[73] (Video 1G). In patients with typically advanced parkinsonism, palilalia may also be observed either irrespective of their medication status[74] or in association with peak doses of levodopa[75] and as a side effect of bilateral stereotaxic thalamotomy, most likely as the effect of lesions.[76] Focal brain lesions, typically affecting thalamic and/or midbrain structures, may also lead to the expression of palilalic behaviors.[34,60,62,76-85] A family with extensive intracerebral calcification was reported to present palilalia,[86] and indeed patients with Fahr syndrome, an etiologically heterogeneous disorder,[87] will often present this clinical sign. Further, palilalia was reported as ictal,[88] autoimmune,[89] and drug-induced phenomenon (eg, with clozapine[90] or cefepime[91]).

Like klazomania, palilalia was also observed in patients with encephalitis lethargica and postencephalitic parkinsonism.[62,64,92-94] Other reported etiologies are early-onset schizophrenia[95] and membranous lipodystrophy.[96] Finally, palilalic behaviors may also be encountered in functional neurological disorders[52] and in culture-bound startle syndromes (eg, Indonesian Latah; also see below).[97] Of note, palilalia should be distinguished from stuttering, a disorder with dysfluency of speech and repetition of sounds, syllables, or words (eg, Video 1A, palilalia vs Video 1K, stuttering).[98,99]

Echolalia

Echolalia is the automatic imitative repetition of sounds, words, or phrases in the absence of explicit awareness.[100] Although echolalia constitutes a physiological neurodevelopmental phenomenon, its unremitting persistence or reemergence may point to pathology.[100]

As with the majority of involuntary vocalizing behaviors, the prevalence and exact characteristics of echolalia in different disorders remain understudied.[101] However, it is most commonly reported in TS[66,102] and ASD.[103-106] Patients with other neurodevelopmental disorders,[15,107-110] including fragile X[108,109] and Williams syndrome,[110] and neurodegenerative syndromes (eg, dementia with Lewy bodies,[111] various tauopathies,[112-117] HD,[118] Creutzfeldt-Jakob disease (CJD),[119] and chorea-acanthocytosis[73]) may also present with echolalia. Neurometabolic disorders, such as Niemann-Pick type C (Video 1L) and Wilson's disease[120] or encephalopathic syndromes,[121-123] as well as brain lesions due to focal or diffuse cerebrovascular damage,[114,124] carbon monoxide poisoning,[60] and severe head trauma[85] were also associated with echolalic behaviors. Infections (eg, cerebral malaria[125]) and autoimmune disorders such as *N*-methyl-D-aspartate (NMDA)–receptor encephalitis,[126] systemic lupus erythematosus,[127] and others[128,129] may also present with echolalia. Drug-induced echolalia was noted with isoniazid,[130] topiramate,[131] ofloxacin,[132] the NMDA-receptor antagonist methoxphenidine,[133] cocaine,[134] designer tryptamine,[135] and phencyclidine ("angel dust," "crystal").[136] Other underlying causes of echolalia are encephalitis lethargica,[94] catatonia,[137] functional neurological disorders (Video 1M[101]), and endemic startle syndromes such as the Jumping Frenchmen of Maine,[138] Latah,[97] and the Ragin' Cajuns of Louisiana.[139] Indeed, in this latter group of etiologies, echolalic behaviors are characteristic.

Coprolalia

The exact definition of coprolalia in the medical context has been tortuous. Essentially, coprolalia denotes the involuntary utterance of obscenities.[52] Intent is an important classifier in coprolalic behaviors, and unfortunately it remains unclear how to objectively distinguish coprolalia from common swearing. In TS, the unintended expression of coprolalic behaviors is encountered in about one-fifth of patients.[140] Typical coprolalic behaviors in TS are characterized by the utterance of single short—in the English language, 4-letter—words with a different pitch or tone from ongoing speech.

There have been only a few reports of patients exhibiting coprolalia in other neurological conditions, such as neurodevelopmental disorders (eg, Kleine-Levin[141] and fragile X syndrome[12]), neurodegenerative syndromes (eg, FTD,[142] Alzheimer's disease,[143] and chorea-acanthocytosis[144]), after focal brain lesions[60], in encephalitis lethargica[94], or as ictal phenomenon.[145] A final category includes functional neurological disorders, and often these patients may be misdiagnosed with TS, although their clinical characteristics may largely differ.[52] Indeed, different from coprolalia in TS, functional coprolalic behaviors often comprise short sentences with obscene content. Most importantly, many of these behaviors are also context dependent (see Video 1N). A previous history of medically unexplained

symptoms and further documented functional neurological signs are typically present.[52]

Vocalizations as Part of Stereotypies

The precise definition of stereotypies and their exact phenomenological distinction from other repetitive motor behaviors, for example, tics, is difficult.[146] The term denotes a repetitive, often continuous, non-goal-directed movement pattern that is typically distractible.[146] As with echolalic behaviors, stereotypies are also part of physiological development that often abate within the first years of life.[146] Although the persistence of stereotypic vocalizations may still be part of normal development,[147] in many cases it signifies pathology, and indeed stereotypic utterances are part of the diagnostic criteria of ASD (Video 2A).[3] One large case series of 83 patients with Rett syndrome described phonic stereotypies with repetitive sounds, words, or phrases in only 6% of patients.[148] We recently observed loud stereotypic vocalizations in a patient with 15q13.3 microdeletion syndrome (Video 2B) and late-treated cases with phenylketonuria. Further, stereotypic vocalizations have been documented in patients with schizophrenia.[149]

Vocalizations as Part of Dystonia, Chorea, and Other Dyskinesias

Involuntary sounds may also be part of dystonic and choreic disorders. For example, lip-smacking sounds (Video 3A) and panting and gasping (Video 3B) are characteristic presentations of drug-related, usually tardive syndromes. Most recently, we documented a case with generalized dyskinetic movements and loud utterances following treatment with lenalidomide (Video 3C). A similar case, albeit without video documentation, was also recently reported.[150] In chorea-acanthocytosis, beyond the presence of tic vocalizations, sounds such as belching, spitting, clicking, sniffing, grunting, sucking, blowing, gasping, sighing, or monosyllabic utterances may be observed.[151] In HD, lip-smacking and grunting (also see below) are frequently reported. In a large cohort of patients with Sydenham's chorea, 8% presented with simple vocalizations (tongue clicking, throat clearing, sniffing) not preceded by premonitory sensations, but in association with facial chorea in most of the patients.[152] It was proposed that the sounds are generated by involuntary choreic activation of pharyngeal and laryngeal muscles.[152]

Continuous Vocalizations Such as Groaning, Moaning, Grunting, and Shrieking

Continuous groaning, moaning, grunting, and shrieking are most frequently associated with neurodegenerative diseases. For example, in dementias, such as Alzheimer's disease[153,154] and others[153,154], to include HD, continuous involuntary vocalizations, often labeled as vocally disruptive behavior, are part of a spectrum of behavioral symptoms that correlate with the severity of cognitive impairment[155] and may be exacerbated with emotional arousal (Video 4A). Purposeless noisemaking, for example, groaning or howling in parkinsonism[156] (Video 4B), including PSP[157-159] (Video 4C), may also be encountered. We also documented continuous shrieking in a patient with acquired hepatocerebral degeneration during an episode of acute encephalopathy (Video 4D). Finally, functional neurological disorders may also present with continuous sounds, such as grunting (Video 4E) or shrieking (Video 4F).

Pathological Laughter and Crying

Laughter and crying behaviors that occur detached from emotional content were reported in patients with TS as part of tic behaviors[160] and other neurodevelopmental disorders (eg, Angelman syndrome,[161] partial trisomy 16p,[15] and Rett-like syndromes[162]). However, pathological laughter and crying is most commonly associated with neurodegenerative disorders, such as ALS,[163] FTD,[164] Alzheimer's disease,[165] primary progressive aphasia,[166] multiple system atrophy cerebellar type,[167] CJD,[168] spinocerebellar ataxia (SCA) 17,[169] and HD. Focal brain lesions in cerebrovascular disease,[170-177] traumatic brain injury,[178-180] autoimmune-mediated lesions in disseminated encephalomyelitis[181-183] or drug-induced behavior[184] are additional etiologies. Finally, recurring "automatic" laughter was also reported as part of ictal phenomena (gelastic seizures).[185,186]

Vocalizations Resembling Physiological Reflexes

Typical vocalizations related to physiological reflexes are sniffing, throat clearing, belching, and wheezing, whereby these audible sounds are mediated by supraglottic structures without involvement of the larynx. Sniffing and throat clearing are noises that are frequently encountered as habitual behaviors (eg, throat clearing in concert halls) and as simple vocal tics in patients with TS.[66] Persistent coughing as a vocal tic can be misinterpreted as disease of the upper and lower airways.[187,188] An extraordinary cause of belching was seen in a patient with parkinsonism, who suffered from a disturbance of esophageal motility with consecutive belching during OFF-periods that remitted with levodopa intake.[189] Persistent hiccups were reported after ischemic lesions of the brain stem.[190,191] Sounds such as coughing or throat clearing may also present either as ictal phenomena[192,193] or "seizure-ending signs."[194] Belching in combination with aerophagia was described in a patient following herpes simplex encephalitis.[195] Sniffing, coughing, belching (Video 5A), and hiccup-related sounds (Video 5B) were also documented in functional neurological disorders.[52,196] Other physiological involuntary

vocalizations are "contagious yawning"[197] or groaning during sexual intercourse.[198]

Others

This group encompasses involuntary vocalizations that may not clearly belong in any of the previous categories and may represent distinctive phenomena of specific etiologies. For example, patients with culture-bound startle syndromes, such as Latah (also see section on palilalia), typically vocalize following a loud external stimulus.[97] Patients with functional movement disorders may also show similarly exaggerated stimulus-triggered responses (Video 6). This type of vocalized startling differs from the classic motor startle response in hyperekplexia. In the classical hereditary forms of hyperekplexia, the latency of the stereotypic spread of muscle activation is very short, whereas in the neuropsychiatric forms the latency is longer and includes a secondary phase with vocalization.[199]

Another important category encompasses ictal phenomena. Ictal vocalizations (also see previous sections) may inherently cover the entire tenor of possible sounds and phonemes of humans: from the classic "ictal cry," signifying the beginning of generalized tonic-clonic seizures,[200] over echo-, pali-, and coprolalic[145,201-204] behaviors, to animal noises ("bleating of sheep," barking),[205,206] singing, and humming.[207-209] Of note, weeping, moaning, and coughing may also be encountered in nonepileptic seizures.[200]

A final category includes noisemaking during sleep. In addition to common snoring, which is the result of obstructed air movement in the upper airways leading to vibration of the soft palate and posterior faucial pillars,[210] other sleep-related sounds include strictly expiratory groaning and moaning, known as catathrenia.[211] In neurodegenerative disorders, such as multiple system atrophy (MSA)[212] or SCA17,[213] stridor during sleep is a common feature. In anti-IgLON5 syndromes, a prominent stridor in association with REM sleep behavior disorder (RBD) does frequently occur.[214] RBD itself may also be associated with vocalizations such as laughing, talking, shouting, and swearing. It has been described in TS[215] and autism.[216] Most commonly, however, RBD occurs in neurodegeneration (eg, α-synucleinopathies,[217-220] tauopathies,[219,221,222] and others[223-228]; see Table 1). RBD was also reported as a result of focal brain lesions, particularly within the brain stem following stroke[229-231] or due to tumors,[232] and in autoimmune disorders, such as multiple sclerosis,[233] Guillain-Barré syndrome,[234] and paraneoplastic encephalitis.[235] It has also been described in association with narcolepsy,[236] epilepsy,[237] and posttraumatic stress disorder.[238] Finally, vocalizations during sleep can be related to night terrors[239] or sleep-related hypermotor seizures.[240] Figure 1 provides a diagnostic algorithm on how to etiologically approach the different involuntary vocalizations described here.

Pathophysiology of Involuntary Vocalizations

The physiology of vocalizing behaviors relies on a well-coordinated network of respiratory, laryngeal, and supralaryngeal muscles.[241] The motoneuronal pool underlying the innervation of these motor effectors is widespread between pontine segments of the brain stem (eg, for the control of jaw-closing muscles) over to motor neurons of the upper lumbar spinal cord (eg, innervation of abdominal muscles).[241] The coordination of this extensive neuronal network is accomplished by superordinate neural structures, which control and maintain the different elements of vocalizing behaviors to include vocal reflexes (eg, shrieking or crying as a result of a painful stimulus), imitative vocalizations, and human speech.[242] Extensive research in a wide range of mammals, including humans, has revealed 2 basic networks underlying vocalization behaviors with overlapping output structures.[242] A cingulo-periaqueductal network has been associated with

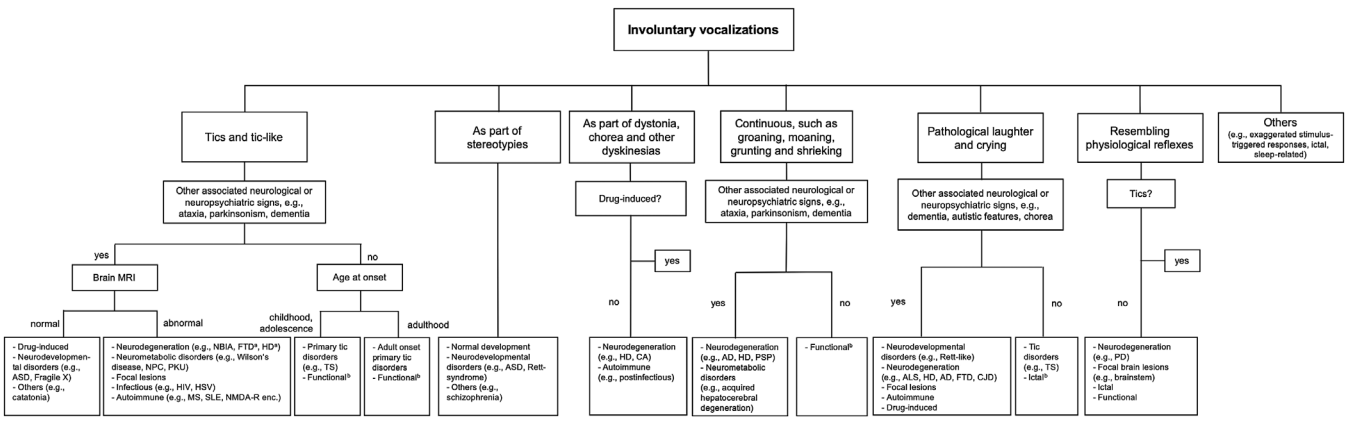

FIG. 1. Diagnostic algorithm for the approach of patients with involuntary vocalizations. [a]MRI might be normal, [b]may also occur with other neuropsychiatric or neurological signs. AD, Alzheimer's disease, ALS, amyotrophic lateral sclerosis, ASD, autism spectrum disorder, CA, chorea-acanthocytosis, CJD, Creutzfeld-Jacob Disease, FTD, frontotemporal dementia, HD, Huntington's disease, MS, multiple sclerosis, NBIA, Neurodegeneration with Brain Iron Accumulation, NPC, Niemann Pick type C, PD, Parkinson's disease, PSP, progressive supranuclear palsy, SLE, systemic Lupus erythematodes, TS, Tourette syndrome. Also refer to table 1 for complete list of etiologies associated with involuntary vocalizations.

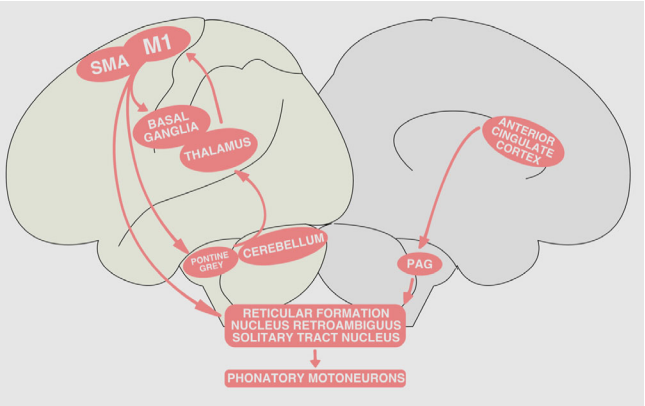

FIG. 2. Simplified representation of the 2 main functional neuroanatomical networks involved in vocalizing behaviors. Left, a motor-cortical-striato-thalamo-cortical network underlying control of learned vocalizations (eg, speaking and singing). Right, a limbic cingulo-periaqueductal network regulating gating control of patterned reflex vocalizations (eg, nonverbal emotional responses, such as crying or moaning). Adapted by references 241 and 242. PAG, periaqueductal gray; SMA, supplementary motor areas; M1, primary motor cortex. [Color figure can be viewed at wileyonlinelibrary.com]

the control of patterned vocalizations related to the gating of reflexes, such as nonverbal emotional responses (eg, crying, moaning, shrieking, and laughing). The supplementary motor area together with the motor cortex, the cortico-striato-thalamo-cortical pathways, and a wider network extending to the pontine gray and cerebellar pathways regulate fine motor control and learned vocalizations, such as the ability to speak and sing. Figure 2 provides a simplified representation of the key neural structures underlying human vocalizing behaviors.

Unfortunately, despite the advances in the field of vocalizations, most pathological phenomena reported here remain scientifically understudied. However, phenomenological observations and cross-species comparative behavioral and neuroanatomical studies, including lesions and chemical and electrical stimulation protocols (reviewed in reference 242) provide important insights into the neuronal structures involved in the different pathologies we present here. Research in tic and tic-like vocalizations implicates 2 key pathophysiological mechanisms for repetitive vocalizations. First, local disinhibition within the cortico-basal ganglia-thalamo-cortical loops that control motor behavior is suggested to lead to amplified output gain. This has been demonstrated in primate and rodent models of tic-like behaviors[243,244] and was further supported by neuropathological studies in patients with TS.[245,246] A single study examined the neuronal locus of disinhibition to produce repetitive grunting sounds, labeled as tic-like behaviors in monkeys, and highlighted the characteristic involvement of the nucleus accumbens and the anterior cingulum, as part of the cingulo-periaqueductal network, underlying these behaviors.[247] As tic vocalizations range from simple nonverbal utterances, such as sniffs or grunts, to words and complete sentences, it is likely that structures of both the cortico-striato-thalamo-cortical and the limbic cingulo-periaqueductal networks are involved in the generation of vocal tics. In turn, pathologically increased output gain, including vocalizations, is further selectively reinforced through enhanced stimulus-response learning via dopaminergic input — here, vocalizing tics receive behavioral salience.[248] The efficacy of antidopaminergic medication (also see below) to treat tic vocalizations corroborates the pathophysiological role of reinforcement learning. Most importantly, disinhibition and enhanced reinforcement learning may either be the result of a neurodevelopmental disorder, as in primary tic disorders, or due to brain damage, as in frontal lobe syndromes or neurodegeneration (also see Tics and Tic-Like Vocalizations section).

It remains unclear why in certain conditions, as in HD, for example, vocal tics such as grunting may often be very specific. In one account, the most commonly employed motor programs would also have the highest probability of being part of tic behaviors. For example, in primary tic disorders, patients mostly exhibit their tics at the motor effectors, which they most commonly employ in their daily living (eg, blinking). In light of the phenomenological overlap between choreic involuntary vocalizations, which may also lead to expiratory gasping, sniffing, or grunting, this view predicts that patients with choreic grunting would also have a high probability of developing grunting tics. Indeed, a clear clinical distinction between choreic grunts and grunting tics may in many cases be notoriously difficult (Video 1E,F vs Video 1G).

Different to tics, vocalizations as part of stereotypies remain less well explored. Certain clinical facts, as for example the absence of a premonitory urge in stereotypies and their typically continuous nature, imply distinct functional neuroanatomical correlates, even though the cortico-basal ganglia-thalamo-cortical circuitry has also been involved.[249-251] The fact that stereotypies can frequently be observed in both humans and animals during confinement and sensory isolation also highlights the significance of self-stimulation in their emergence and maintenance.[252,253]

The pathophysiology of involuntary sounds as part of dystonia, chorea, and other dyskinesias, is intrinsically related to the nature of the involuntary movements[254-256] and is beyond the scope of this article. Indeed, the vocalizing sounds are the result of involuntary activation of structures related to the respiratory and vocal apparatus, but do not, we posit, involve higher-order neural processes that produce patterned behaviors such as speech. Beyond the few neurodegenerative choreic disorders we have included, most syndromes we have identified are drug-induced, and indeed extensive literature exists about the pathophysiology of drug-induced movement disorders, including vocalizations (for example, reviewed in references [257] and [258]). Video 3C demonstrates lenalidomide-induced vocalizations as part of a choreodystonic syndrome. Only one similar case has been

documented previously.[150] Although the exact mechanism of action remains unclear, we do wish to note the unusual and dramatic side effect of this medication.[259]

One particular category includes continuous vocalizations, including groaning, moaning, grunting, and shrieking. We previously published a case (Video 4C) in which we highlighted the role of distinct neural generators in vocalizing behaviors. We postulated that continuous groaning could be the result of ongoing activation of the cingulo-periaqueductal circuit, described above (also see Fig. 2), as a result of either enhanced excitation, reduced top-down inhibition, or both.[159] Given the common denominator of many of the disorders we report here linked to frontal lobe damage, we suggest that loss of inhibitory control over a subcortical cingulo-periaqueductal circuit involved in the generation of nonverbal utterances could lead to these types of behaviors.[159,242] Additional factors, such as enhanced limbic drive and dysfunction of the serotonergic system,[260] may further strengthen and/or perpetuate these behaviors. We suggest that the pathophysiology of pathological laughter and crying also falls within this pathophysiological category — with the exception of gelastic seizures as ictal phenomena, which are typically associated with hypothalamic hamartomas.[261,262] Epileptic activity of the frontal lobes, including the anterior cingulate cortex,[263-266] but also parietal[267] and temporal lobes,[268] has also been reported to give rise to gelastic seizures.

Within the group of vocalizations and sounds that resemble physiological reflexes, the most common etiologies are indeed tics (also see above) and functional neurological disorders. The pathophysiology of functional neurological disorders, including movement disorders, has been reviewed before.[269-271] It is important to note that tics, vocalizations as part of stereotypies and vocalizations as part of a functional disorder, are typically distractible. This highlights that for these particular vocalizing behaviors, superintending centers related to attention and potentially motivation can alter the output gain based on environmental context.

In ictal vocalizations, the behavioral abnormality depends on the cortical locus of abnormal neuronal excitation. For example, seizures over the temporal lobe typically elicit various types of different vocalizations, such as animal noises, coprolalia, throat clearing, and belching.[192,193,201,205] Similar vocalization behaviors have also been described for epileptic discharges over mesiofrontal brain areas, including the supplementary motor area and the anterior cingulate cortex.[242,272]

Finally, vocalizations in REM sleep disorder are suggested to result from dysfunction of the nucleus subcoeruleus and/or the reticular formation, whose glutamatergic, GABAergic, and glycinergic projections fail to inhibit spinal motor neurons, and thus muscle atonia is no longer induced.[273] Neurodegenerative disorders, such as PD,[274] MSA, DLB, and PSP,[275] with abnormalities in REM sleep behavior typically affect these structures, and indeed the reticular formation is a key structure for the activation of the motor neuronal pool involved in vocalizations (Fig. 2).

Treatment Options

Within the range of the different involuntary vocalizations, the treatment strategy depends on the vocalization type and the underlying etiology. However, beyond the treatment of tics, therapeutic interventions in other types of vocalizations are mostly based on case series and single case reports. For tic vocalizations, as in the example of primary tic disorders, there are 3 main therapeutic venues: (1) behavioral treatments, including habit reversal training and its expansion, the comprehensive behavioral intervention for tics (CBIT) (for a review, see reference 276; (2) pharmacological interventions, such as antipsychotics, dopamine-depleting agents, α_2-agonists, and more recently cannabinoids[277-279] (3) surgical interventions for refractory cases, such as deep brain stimulation.[280] In addition, local injections of botulinum toxin might also alleviate symptoms.[281] Single case reports have indicated that other medications might also be helpful. For example, fluoxetine was used to control laughing tics in TS.[160] However, the efficacy of these treatments remains understudied. An important caveat is the treatment of tic-like behaviors in functional neurological disorders in which behavioral therapies should be preferred over pharmacological agents.[50-52,54,282]

In klazomania, particularly in the presence of depression and anxiety, benzodiazepines showed some therapeutic promise in 1 case, whereas quetiapine, risperidone, aripiprazole, amitriptyline, and sertraline were ineffective.[57] Electroconvulsive therapy was also reported to be effective in 2 patients with klazomania and depression.[57,58]

Treatment reports specifically targeting pali-, echo-, and coprolalia are particularly rare. Palilalia in vascular dementia was responsive to the antidepressant trazodone.[283] In some cases of echo- and coprolalia, benzodiazepines led to the alleviation of symptoms.[94,123] The amphetamine-related drug fenfluramine was efficient in the reduction of echolalia in 10 patients with ASD.[107] Echolalia in a case with a left temporoparietal hemorrhage and a case with a diagnosis of Rubinstein-Taybi syndrome improved after behavioral therapy.[107,284] We believe that behavioral therapy should be a first-line option in patients with repetitive vocalizing behaviors, such as pali-, echo-, or coprolalia, but also in cases with vocalizations as part of stereotypies. However, in some of these cases, particularly in the presence of additional behavioral abnormalities, pharmacological augmentation may be necessary.

In vocalizations as part of dystonia, chorea, or other dyskinesias, the most common etiology is drug-induced. In these cases, the causing agent should be removed if possible, or dosage should be reduced. In addition, the prescription of dopamine-depleting agents might be helpful.[2]

TABLE 2. Treatment options for involuntary vocalizations

Vocalization	Treatment option
Tics and tic-like vocalizations	Behavioral therapy — HRT, CBIT — ERP Pharmacological treatments — Antipsychotics (eg, aripiprazole, risperidone, olanzapine) — α2-Agonists (eg, clonidine, guanfacine) — Dopamine-depleting drugs (eg, tetrabenazine) — Cannabinoids — Botulinum toxin — Others (eg, baclofen, topiramate) DBS Electroconvulsive therapy (for klazomania)[a]
Vocalizations as part of stereotypies	Behavioral therapy (eg, CBT) Pharmacological treatments — Antipsychotics (eg, haloperidol, risperidone, olanzapine) — Antidepressants (eg, SSRI such as fluoxetine, SNRI such as sertraline, citalopram) — Botulinum toxin
Vocalizations as part of chorea, dystonia, and other dyskinesias	Pharmacological therapy — Reduce offending agent if possible — Dopamine-depleting drugs (eg, tetrabenazine, deutetrabenazine)
Continuous vocalizations such as groaning, moaning, grunting, and shrieking	Behavioral therapy — Practical interventions (eg, relieve discomfort, provide orientation, avoid excess attention to vocalizing behavior) Pharmacological therapy — Benzodiazepines (eg, lorazepam) — Antipsychotics (eg, risperidone) — Antidepressants (eg, tricyclic such as doxepin, SSRI such as paroxetine, citalopram, SARI such as trazodone) — β-blockers (eg, propranolol)
Pathological laughter and crying	Pharmacological therapy — Antidepressants (eg, tricyclic such as doxepin, SSRI such as paroxetine, citalopram, and other such as venlafaxine, mirtazapine, or reboxetine) — Dopaminergic drugs (eg, levodopa) — NMDA-receptor antagonists
Vocalizations resembling physiological reflexes	Behavioral therapy

CBIT, comprehensive behavioral intervention for tics; CBT, cognitive behavioral therapy; DBS, deep brain stimulation; ERP, exposure and response prevention; HRT, habit reversal training; NMDA, *N*-methyl-D-aspartate; SARI, serotonin antagonist and reuptake inhibitor; SNRI, serotonin-norepinephrine reuptake inhibitor; SSRI, selective serotonin reuptake inhibitor.
[a]Two case reports.

Although being a huge burden in hospitals and nursing homes, specific treatment for continuous vocalizing behaviors, such as those associated with neurodegeneration, is poorly investigated. A detailed assessment about whether other circumstances such as physical or mental suffering (pain, discomfort, fatigue, frustration, depressed mood, deprivation, etc.) could elicit or precipitate the vocalizing behavior is recommended. The recognition and removal of these factors could lead to a remission of vocalizing behaviors. In addition, behavioral interventions such as avoidance of positive reinforcement of vocally disruptive behavior could be helpful.[285] Pharmacological approaches include tranquilizers, antipsychotics, anticonvulsants, antidepressants, and beta-blockers, however, with mixed responses.[154] In the absence of randomized, controlled studies, the antidepressants paroxetine,[286] citalopram,[287] trazodone,[288,289] and doxepine[290] were shown to reduce vocalizing behavior in single cases and case series. Although reported to be the most effective,[155] benzodiazepine[285] intake should be monitored with caution to maintain functionality and mobility. In cases with concomitant aggression, antipsychotic medication could be helpful, and in patients with comorbid depression or anxiety, the usage of antidepressants is preferable.[285] Pathological crying after brain injury was reported to be well controlled with paroxetine and citalopram in a large case series.[291] Intractable hiccups responded well to inhaled cannabis in a patient with AIDS.[292] Table 2 provides a comprehensive overview of treatment options in involuntary vocalizations.

Conclusion

We here presented the wide range of involuntary vocalizations in humans, together with 29 video-documented cases to exemplify their phenomenology. Based on these cases and on the extensive literature review, we provide a diagnostic algorithm to guide clinicians in approaching patients with involuntary vocalizing behaviors (Fig. 1), discuss their pathophysiology, and provide treatment options, where available. We do recognize that some of the behaviors that we document reflect sounds emitted from supraglottic structures, rather than true vocalizations generated from the vocal cords, and have clearly documented the differences between these phenomena. Also, we are aware that the classification of some of the vocalizations we present as involuntary (eg, tics) may be open to criticism. However, we do suggest that several of their qualities, for example, their inflexible, repetitive, and socially inopportune character, as well as their perception as unwanted and often distressing phenomena, guarantee a minimal involuntary component. Our algorithmic approach may not cover every possible clinical presentation of involuntary vocalizations and its respective etiology. Nevertheless, we do hope that it provides a clear framework to guide clinicians in their diagnostic considerations. This, in turn, will translate to improved pathophysiological understanding and appropriate management of these paradigmatic neuropsychiatric patients.

Legends to the Videos

Video 1. Tics and tic-like vocalizations. (A–C) Vocalizations in TS. (A) Multiple vocal tics including whistling,

grunting, sighing, palilalia ("ja, ja, hallo, hallo, hallo," ie, "yes, yes, hello, hello, hello") and coprolalia ("scheiße"). Motor (facial twitches) and vocal tics (humming) started at age 12. Tics were preceded by premonitory urges and were suppressible on demand. The patient was also diagnosed with obsessive-compulsive disorder, attention deficit hyperactivity disorder, depression, and anxiety disorder. (B) Tic vocalizations including nonsensical sounds, words ("der Kampf") and phrases ("Hilfe, L… stirbt") including coprolalia. Tic behaviors first appeared at age 5, waxed and waned over time, were preceded by premonitory urges, and could be voluntarily suppressed. (C) Bout of grunting, throat clearing, and coughing tics in a patient with TS. Motor and vocal tics were present since the ages of 5 and 12 years, respectively, and waxed and waned with time. Tics were preceded by premonitory urges and could be voluntarily suppressed. Severe obsessive-compulsive and major depressive disorder were also diagnosed. (D–F) Vocalizations in HD. (D) Shrieking, sniffing and shouting tics. (E) Characteristic repetitive grunting tics and sniffing sounds. The patient described a mounting urge sensation in his larynx preceding and leading to the release of these sounds. (F) Grunting, throat clearing, and coughing tics (previously published[293]). The involuntary phenomena could be suppressed for a few seconds until an unpleasant tension and tightness led to their continuation. (G) Laughter, rasping sounds, grunting, hissing, snorting, and palilalic utterance of nonsensical words ("upsa") in monozygotic twins with chorea-acanthocytosis (previously published without video material[20]). (H) Drug-induced (risperidone and methylphenidate overdose) lip-smacking tics in a patient with schizophrenia. He was able to briefly voluntarily suppress the repetitive lip-smacking movements but experienced an increasing urge to release them. Treatment with tetrabenazine improved the repetitive behaviors. (I) Involuntary shouting (klazomania) in a patient with TS. Eye blinking was the first tic at age 10, followed by multiple waxing and waning motor and vocal tics. Over 2 years the patient presented a complex pattern of motor and vocal tics with repetitive foot stamping, flailing movements of the arms, and grimacing alongside bouts of loud shouting. Severe obsessive-compulsive disorder and self-injurious behavior (hitting his head, pressing against his eye, scratching) were also present. (J) Recurrent shouting (klazomania) in a patient with functional disorder (previously published without video material[294]). The patient first developed sudden jerks of the head, neck, and left arm combined with involuntary vocalizations such as screams, yelps, and grunts a few days after a minor traffic accident at age 33. Sudden movements and screams were not preceded by premonitory urges, were not suppressible, and were triggered by unexpected bright lights or taps, stress, and anger, but also occurred spontaneously. Neurophysiological analysis of startle-induced behaviors showed variable patterns of muscle activation and prolonged activation latencies. (K) Stuttering in a patient with Parkinson's disease and deep brain stimulation (DBS) in DBS-OFF (K-1) and DBS-ON (K-2) conditions. (L) Echolalia ("mit mir," ie, "with me") in a patient with Niemann-Pick type C. (M) Echolalia ("Christmas," "ice cream," "bugger") in a patient with a functional neurological disorder. She presented with jerks, which first started in her right arm during a driving lesson 2 years earlier and then spread over her whole body. During the same period, she began to repeat words spoken by other people (echolalia) and imitate other people's actions (echopraxia). Movements and vocalizations, although sometimes preceded by inner tension, could not completely be inhibited voluntarily. However, they were distractible. Sudden spontaneous jerking during walking was also documented (previously published[295]). (N) Repetitive continuous swearing ("functional coprolalia") in a patient with functional neurological disorder and a previous diagnosis of TS. The repetitive swearing ("Hure") occurred in bouts and over prolonged periods and was context dependent, that is, triggered only when the patient met his previous partner or discussed her. During the same period, he also developed a functional gait disorder, which he described as the inability to walk as a result of "extreme tension" that lasted for a period of 2 years and resolved spontaneously.

Video 2. Vocalizations as part of stereotypies. (A) Stereotypic vocalizations accompanied by motor stereotypies (repetitive touching of the right ear) in a patient with autism spectrum disorder, before (A-1), during (A-2), and after (A-3) treatment with botulinum toxin of the vocal cords. (B) Stereotypic shouts accompanied by motor stereotypies (flexion-extension movement of the upper extremity) in a patient with 15q13.3 microdeletion syndrome and cognitive disability, impulsivity, short stature, cachexia, and mitral valve insufficiency. The stereotypic behavior developed 4 years earlier during a stressful period. The patient reported a soothing character of the repetitive shouts and movements, which reduced a feeling of inner distress. The behavior was distractible, although the patient felt that she was not able to suppress the movements and vocalizations.

Video 3. Vocalizations as part of chorea, dystonia, and other dyskinesias. (A) Lip smacking in a patient with tardive dyskinesia. (B) Panting and gasping in a patient with tardive dyskinesia due to chronic metoclopramide intake. (C) Acute-onset hissing and shrieking in a patient with generalized choreodystonia subsequent to lenalidomide treatment for multiple myeloma.

Video 4. Continuous groaning, moaning, grunting, and shrieking. (A) Repetitive shouting associated with emotional discomfort in a patient with HD. (B) Continuous howling in a patient with parkinsonism and dementia. (C) Continuous groaning in a patient with PSP

(previously published[159]). (D) Continuous shrieking in a patient with acquired hepatocerebral degeneration during an acute encephalopathic episode (D-1) and after treatment (D-2). (E) Continuous grunting with distractibility (E-1) in a patient with a functional neurological disorder. The continuous vocalizing behavior, which was distractible and entrainable, began abruptly 4 years ago and was perceived as involuntary. It remitted during talking, eating, and drinking. (F) Continuous shrieking in a patient with functional neurological disorder (previously published without video material[294]). Repetitive inspiratory shrieking associated with facial grimacing, eye closure, and variable jerks of the head and upper extremities triggered by unexpected, loud noises or also occurring spontaneously. These behaviors, which were not preceded by premonitory urges and were not suppressible, appeared 1 year after a head injury as a result of a traffic accident. Comorbid anxiety disorder with panic attacks and forgetfulness were noted.

Video 5. Vocalizations resembling physiological reflexes. (A) Air gasping and belching as a result of functional aerophagia in a patient with functional neurological disorder. She described suffering from anxiety episodes, which led to aerophagic behaviors with subsequent gastric distention and belching. (B) Recurrent hiccup-like sounds in a patient with functional neurological disorder. These appeared abruptly following an episode of severe diarrhea after food poisoning. She could voluntarily suppress the hiccup-like sounds by bending over or pressing the arms against the abdominal wall, but otherwise felt that she had no control over them. Hiccup-like vocalizations remitted during eating and drinking.

Video 6. Other involuntary vocalizations. Exaggerated pseudo-startle response with shouting and hissing in a patient with functional neurological disorder. There was a variable pattern of muscle recruitment and vocalizations throughout examination, following acoustic and light tactile stimuli over different body areas, but also preceding those. An irregular and frequency-variable tremor of both arms, which was distractible and entrainable, was also noted. "Huffing and puffing" and other effortful behaviors were documented during neurological examination.

Acknowledgments: The authors thank Dr. Andreas Horn for his support in preparing the medical illustration.

References

1. Fitch WT. The evolution of speech: a comparative review. Trends Cogn Sci 2000;4(7):258–267.
2. Tolosa E, Peña J. Involuntary vocalizations in movement disorders. Adv Neurol 1988;49:343–363.
3. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 5th edition. Arlington, VA: American Psychiatric Publishing; 2013.
4. Ganos C, Martino D. Tics and Tourette syndrome. Neurol Clin 2015;33(1):115–136.
5. Kwak C, Dat Vuong K, Jankovic J. Premonitory sensory phenomenon in Tourette's syndrome. Mov Disord 2003;18(12):1530–1533.
6. Leckman JF, Walker DE, Cohen DJ. Premonitory urges in Tourette's syndrome. Am J Psychiatry 1993;150(1):98–102.
7. Reese HE, Scahill L, Peterson AL, et al. The premonitory urge to tic: measurement, characteristics, and correlates in older adolescents and adults. Behav Ther 2014;45(2):177–186.
8. Nass R, Gutman R. Boys with Asperger's disorder, exceptional verbal intelligence, tics, and clumsiness. Dev Med Child Neurol 1997; 39(10):691–695.
9. Stern JS, Robertson MM. Tics associated with autistic and pervasive developmental disorders. Neurol Clin 1997;15(2):345–355.
10. Kahl U, Schunke O, Schöttle D, et al. Tic Phenomenology and Tic Awareness in Adults With Autism. Mov Disord Clin Pract 2015; 2(3):237–242.
11. Jensen E, Palacios E, Drury S. Klinefelter's Syndrome in a 5-Year-Old Boy with Behavioral Disturbances and Seizures. Psychosomatics 2011;52(6):575–578.
12. Schneider SA, Robertson MM, Rizzo R, Turk J, Bhatia KP, Orth M. Fragile X syndrome associated with tic disorders. Mov Disord 2008;23(8):1108–1112.
13. Hassiem F, Cavanna AE. Multiple tics in a patient with Adams-Oliver syndrome. J Neuropsychiatry Clin Neurosci 2015;27(1):e80.
14. Taylor LD, Krizman DB, Jankovic J, et al. 9p Monosomy in a patient with Gilles de la Tourette's syndrome. Neurology 1991; 41(9):1513–1515.
15. Hebebrand J, Martin M, Körner J, et al. Partial trisomy 16p in an adolescent with autistic disorder and Tourette's syndrome. Am J Med Genet 1994;54(3):268–270.
16. S-S, Ren R-J, Wang Y, Wang G, Chen S-D. Tics as an initial manifestation of juvenile Huntington's disease: case report and literature review. BMC Neurology [Internet]. 2017 Dec http://bmcneurol.biomedcentral.com/articles/10.1186/s12883-017-0923-1. Accessed February 4, 2019.
17. Kerbeshian J, Burd L, Leech C, Rorabaugh A. Huntington disease and childhood-onset Tourette syndrome. Am J Med Genet 1991; 39(1):1–3.
18. Jankovic J, Ashizawa T. Tourettism associated with Huntington's disease. Mov Disord 1995;10(1):103–105.
19. Benninger F, Afawi Z, Korczyn AD, et al. Seizures as presenting and prominent symptom in chorea-acanthocytosis with c.2343del *VPS13A* gene mutation. Epilepsia 2016;57(4):549–556.
20. Müller-Vahl KR, Berding G, Emrich HM, Peschel T. Chorea-acanthocytosis in monozygotic twins: clinical findings and neuropathological changes as detected by diffusion tensor imaging, FDG-PET and (123)I-beta-CIT-SPECT. J Neurol 2007;254(8):1081–1088.
21. Dulski J, Sołtan W, Schinwelski M, et al. Clinical variability of neuroacanthocytosis syndromes-a series of six patients with long follow-up. Clin Neurol Neurosurg 2016;147:78–83.
22. Borghero G, Floris G, Cannas A, et al. A patient carrying a homozygous p.A382T TARDBP missense mutation shows a syndrome including ALS, extrapyramidal symptoms, and FTD. Neurobiol Aging 2011;32(12):2327.e1–2327.e5.
23. Rojo A, Pernaute RS, Fontán A, et al. Clinical genetics of familial progressive supranuclear palsy. Brain 1999;122(Pt 7):1233–1245.
24. da Costa RQM, Marrocos RP, Leite MAA, Porto FHG. All that glitters is not gold: When motor and vocal tics in a child do not match Tourette syndrome: A case report. Dement Neuropsychol 2016;10(3):251–253.
25. Arruda WO, Munhoz RP, de Bem RS, et al. Pathogenic compound heterozygous ATP7B mutations with hypoceruloplasminaemia without clinical features of Wilson's disease. J Clin Neurosci 2014; 21(2):335–336.
26. Bilder DA, Kobori JA, Cohen-Pfeffer JL, Johnson EM, Jurecki ER, Grant ML. Neuropsychiatric comorbidities in adults with phenylketonuria: A retrospective cohort study. Molecular Genetics and Metabolism 2017;121(1):1–8.

27. Kumar R, Lang AE. Tourette syndrome. Secondary tic disorders. Neurol Clin 1997;15(2):309–331.
28. Krauss JK, Jankovic J. Tics secondary to craniocerebral trauma. Mov Disord 1997;12(5):776–782.
29. Alioglu Z, Boz C, Sari A, Aynaci M. Transient tic disorder following carbon monoxide poisoning. J Neuroradiol. 2004;31(3): 231–233.
30. Jung N-Y, Lee J-H. Secondary tics after osmotic demyelination syndrome involving both the striatum and the cerebral cortex. J Clin Neurosci 2012;19(1):179–180.
31. Chemali Z, Bromfield E. Tourette's syndrome following temporal lobectomy for seizure control. Epilepsy Behav 2003;4(5):564–566.
32. Singer HS, Dela Cruz PS, Abrams MT, Bean SC, Reiss AL. A Tourette-like syndrome following cardiopulmonary bypass and hypothermia: MRI volumetric measurements. Mov Disord 1997; 12(4):588–592.
33. Yochelson MR, David RG. New-onset tic disorder following acute hemorrhage of an arteriovenous malformation. J Child Neurol 2000;15(11):769–771.
34. Dale RC, Church AJ, Heyman I. Striatal encephalitis after varicella zoster infection complicated by Tourettism. Mov Disord 2003; 18(12):1554–1556.
35. Northam RS, Singer HS. Postencephalitic acquired Tourette-like syndrome in a child. Neurology 1991;41(4):592–593.
36. McDaniel JS, Summerville MB. Tic disorder associated with encephalopathy in advanced HIV disease. Gen Hosp Psychiatry 1994;16(4):298–300.
37. Mejia NI, Jankovic J. Secondary tics and tourettism. Braz J Psychiatry 2005;27(1):11–17.
38. Nociti V, Fasano A, Bentivoglio AR, et al. Tourettism in multiple sclerosis: a case report. J Neurol Sci 2009;287(1–2):288–290.
39. Mrabet S, Benrhouma H, Kraoua I, et al. Mixed movements disorders as an initial feature of pediatric lupus. Brain Dev 2015;37(9): 904–906.
40. Budman C, Sarcevic A. An Unusual Case of Motor and Vocal Tics With Obsessive-Compulsive Symptoms in a Young Adult With Behcet's Syndrome. CNS Spectr 2002;7(12):878–881.
41. Martino D, Chew N-K, Mir P, Edwards MJ, Quinn NP, Bhatia KP. Atypical movement disorders in antiphospholipid syndrome: Atypical Movement Disorders. Mov Disord 2006;21(7): 944–949.
42. Sotero de Menezes MA, Rho JM, Murphy P, Cheyette S. Lamotrigine-induced tic disorder: report of five pediatric cases. Epilepsia 2000;41(7):862–867.
43. Lombroso CT. Lamotrigine-induced tourettism. Neurology 1999; 52(6):1191–1194.
44. Kurlan R, Kersun J, Behr J, et al. Carbamazepine-induced tics. Clin Neuropharmacol. 1989;12(4):298–302.
45. Kayhan F, Uguz F, Kayhan A, Toktaş FI. Bupropion XL-induced motor and vocal tics. Clin Neuropharmacol 2014;37(6):192–193.
46. Pascual-Leone A, Dhuna A. Cocaine-associated multifocal tics. Neurology 1990;40(6):999–1000.
47. Yogaratnam J, Xu C, Thinn DSS, Yoong LK, Khoo CL, Sim K. De novo Tardive Tourette-like syndrome after prolonged combination depot and oral neuroleptic therapy. Acta Neuropsychiatr. 2013; 25(2):122–124.
48. Fountoulakis KN, Samara M, Siapera M, Iacovides A. Tardive Tourette-like syndrome: a systematic review. Int Clin Psychopharmacol 2011;26(5):237–242.
49. Lal S, AlAnsari E. Tourette-like syndrome following low dose short-term neuroleptic treatment. Can J Neurol Sci 1986;13(2): 125–128.
50. Baizabal-Carvallo JF, Jankovic J. The clinical features of psychogenic movement disorders resembling tics. J Neurol Neurosurg Psychiatry 2014;85(5):573–575.
51. Demartini B, Ricciardi L, Parees I, Ganos C, Bhatia KP, Edwards MJ. A positive diagnosis of functional (psychogenic) tics. Eur J Neurol 2015;22(3):527-536.
52. Ganos C, Edwards MJ, Müller-Vahl K. "I swear it is Tourette's!": On functional coprolalia and other tic-like vocalizations. Psychiatry Res 2016;246:821–826.
53. Ganos C, Müller-Vahl K. Cannabinoids in functional tic-like movements. Parkinsonism Relat Disord 2019;60:179–181.
54. Ganos C, Martino D, Espay AJ, Lang AE, Bhatia KP, Edwards MJ. Tics and functional tic-like movements: can we tell them apart? Neurology. Epub 2019 Sep24. https://doi.org/10.1212/WNL.0000000000008372.
55. Benedek L. Zwangsmäßiges Schreien in Anfällen als postencephalitische Hyperkinese. Zeitschrift für die gesamte, Neurologie und Psychiatrie 1925;98:17–26.
56. Comings DE, Comings BG. A controlled study of Tourette syndrome. IV. Obsessions, compulsions, and schizoid behaviors. Am J Hum Genet 1987;41(5):782–803.
57. Bourgeois JA, Li D, Hategan A. Recurrent Klazomania Responsive to Acute Plus Maintenance Electroconvulsive Therapy, J ECT. 2019;35(1):e2–e3.
58. Hategan A, Bourgeois JA. Compulsive shouting (klazomania) responsive to electroconvulsive therapy. Psychosomatics 2013; 54(4):402–403.
59. Pillai P, Krishna N, Regenold W. Klazomania: A Rare Case of Compulsive Screaming, Complicating Major Depression, Effectively Treated with Electroconvulsive Therapy (ECT). J Aging Sci [Internet]. 2017. https://www.esciencecentral.org/journals/klazomania-a-rare-case-of-compulsive-screaming-complicating-majordepression-effectively-treated-with-electroconvulsive-therapy-ect-2329-8847-1000170.php?aid=85506.
60. Pulst S-M, Walshe TM, Romero JA. Carbon Monoxide Poisoning With Features of Gilles de la Tourette's Syndrome. Arch Neurol 1983;40(7):443–444.
61. Bates GDL, Lampert I, Prendergast M, Van Woerkom AE. Klazomania: The screaming tic. Neurocase 1996;2(1):31–34.
62. Critchley M. ON PALILALIA. J Neurol Psychopathol 1927;8(29): 23–32.
63. Oliver WA. Palilalia. Cal West Med 1934;41(5):328–330.
64. Sterling W. Palilalie et le symptom linguosalivaire dans le parkinsonisme encephalitique. Rev Neurol (Paris) 1924;(1):205–220.
65. Micheli F, Gatto M, Gershanik O, Steinschnaider A, Fernandez Pardal M, Massaro M. Gilles de la Tourette syndrome: clinical features of 75 cases from Argentina. Behav Neurol 1995;8(2):75–80.
66. Cardoso F, Veado CC, de Oliveira JT. A Brazilian cohort of patients with Tourette's syndrome. J Neurol Neurosurg Psychiatry 1996;60(2):209–212.
67. Sambrani T, Jakubovski E, Müller-Vahl KR. New Insights into Clinical Characteristics of Gilles de la Tourette Syndrome: Findings in 1032 Patients from a Single German Center. Fron Neurosci 2016;10:415.
68. Stribling P, Rae J, Dickerson P. Two forms of spoken repetition in a girl with autism. Int J Lang Commun Disord 2007;42(4): 427–444.
69. Kluin KJ, Foster NL, Berent S, Gilman S. Perceptual analysis of speech disorders in progressive supranuclear palsy. Neurology 1993;43(3 Pt 1):563–566.
70. Testa D, Monza D, Ferrarini M, Soliveri P, Girotti F, Filippini G. Comparison of natural histories of progressive supranuclear palsy and multiple system atrophy. Neurol Sci 2001;22(3):247–251.
71. Hier DB, Hagenlocker K, Shindler AG. Language disintegration in dementia: effects of etiology and severity. Brain Lang 1985;25(1): 117–133.
72. Papadimas GK, Paraskevas GP, Zambelis T, et al. The multifaceted clinical presentation of VCP-proteinopathy in a Greek family. Acta Myol 2017;36(4):203–206.
73. Saiki S, Hirose G, Sakai K, et al. Chorea-acanthocytosis associated with tourettism: Clinical/Scientific Notes. Mov Disord 2004;19(7): 833–836.
74. Benke T. Repetitive speech phenomena in Parkinson's disease. J Neurol Neurosurgery Psychiatry 2000;69(3):319–324.

75. Ackermann H, Ziegler W, Oertel WH. Palilalia as a symptom of levodopa induced hyperkinesia in Parkinson's disease. J Neurol Neurosurg Psychiatry 1989;52(6):805–307.
76. Stracciari A, Guarino M, Cirignotta F, Pazzaglia P. Development of palilalia after stereotaxic thalamotomy in Parkinson's disease. Eur Neurol 1993;33(3):275–276.
77. Kwon M, Lee J-H, Kim J, Kim JS. Hypokinetic dysarthria and palilalia in midbrain infarction. J Neurol Neurosurg Psychiatry 2008;79(12):1411–1412.
78. Gorno ML, Miozzo A, Mattioli F, Cappa SF. Isolated palilalia: a case report. Eur J Neurol 1997;4(1):94–96.
79. Yasuda Y, Akiguchi I, Ino M, Nabatabe H, Kameyama M. Paramedian thalamic and midbrain infarcts associated with palilalia. J Neurol Neurosurg Psychiatry 1990;53(9):797–799.
80. Horner J, Massey EW. Progressive dysfluency associated with right hemisphere disease. Brain Lang 1983;18(1):71–85.
81. Tomic G, Stojanovic M, Pavlovic A, et al. Speech and language disorders secondary to diffuse subcortical vascular lesions: Neurolinguistic and acoustic analysis. A case report. J Neurol Sci 2009;283(1–2):163–169.
82. Serra-Mestres J, Robertson MM, Shetty T. Palicoprolalia: an unusual variant of palilalia in Gilles de la Tourette's syndrome. J Neuropsychiatry Clin Neurosci 1998;10(1):117–118.
83. Dietl T, Auer DP, Modell S, Lechner C, Trenkwalder C. Involuntary vocalisations and a complex hyperkinetic movement disorder following left side thalamic haemorrhage. Behav Neurol 2003; 14(3–4):99–102.
84. Kuoppamäki M, Rothwell JC, Brown RG, Quinn N, Bhatia KP, Jahanshahi M. Parkinsonism following bilateral lesions of the globus pallidus: performance on a variety of motor tasks shows similarities with Parkinson's disease. J Neurol Neurosurg Psychiatry 2005;76(4):482–490.
85. Thomsen IV, Skinhoj E. Regressive language in severe head injury. Acta Neurol Scand 1976;54(3):219–226.
86. Boller F, Boller M, Denes G, Timberlake WH, Zieper I, Albert M. Familial palilalia. Neurology 1973;23(10):1117–1125.
87. Batla A, Tai XY, Schottlaender L, Erro R, Balint B, Bhatia KP. Deconstructing Fahr's disease/syndrome of brain calcification in the era of new genes. Parkinsonism Relat Disord 2017;37:1–10.
88. Landi D, Benvenga A, Quattrocchi CC, et al. Complex epileptic palilalia: a case report. Seizure 2012;21(8):655–657.
89. Patira R, Smith-Benjamin S, Ramachandran VS, Altschuler EL. Palilalia due to steroid-responsive encephalopathy. Neurol Clin Pract 2017;7(3):e23–e25.
90. Azevedo JC, Lopes R, Curral R, Esteves MF, Coelho R, Roma-Torres A. Clozapine-induced palilalia?. Acta Neuropsychiatrica. 2012;24(02):122–124.
91. Tamune H, Hamamoto Y, Aso N, Yamamoto N. Cefepime-induced encephalopathy: Neural mass modeling of triphasic wave-like generalized periodic discharges with a high negative component (Tri-HNC). Psychiatry Clin Neurosci 2019;73(1):34–42.
92. Leyser E. Die zentralen Dysarthrien und ihre Pathogenese. Klinische Wochenschrift 1923;(2):2176–2179.
93. Bálint A, Julius D. Sprachiterationen und Psychose bei Encephalitis epidemica. European Neurology 1925;58(2–3):102–120.
94. Dale RC, Webster R, Gill D. Contemporary encephalitis lethargica presenting with agitated catatonia, stereotypy, and dystonia-parkinsonism. Mov Disord 2007;22(15):2281–2284.
95. Ferrara M, Freda F, Massa R, Carratelli TJ. Frontal lobe syndrome or adolescent-onset schizophrenia? A case report. Acta Psychiatrica Scandinavica 2006;114(5):375–377.
96. Ueki Y, Kohara N, Oga T, et al. Membranous lipodystrophy presenting with palilalia: a PET study of cerebral glucose metabolism. Acta Neurol Scand 2000;102(1):60–64.
97. Bakker MJ, van Dijk JG, Pramono A, Sutarni S, Tijssen MAJ. Latah: an Indonesian startle syndrome. Mov Disord 2013;28(3): 370–379.
98. Tsuboi T, Watanabe H, Tanaka Y, et al. Early detection of speech and voice disorders in Parkinson's disease patients treated with subthalamic nucleus deep brain stimulation: a 1-year follow-up study. J Neural Transm (Vienna) 2017;124(12):1547–1556.
99. Prasse JE, Kikano GE. Stuttering: an overview. Am Fam Physician 2008;77(9):1271–1276.
100. Ganos C, Ogrzal T, Schnitzler A, Munchau A. The pathophysiology of echopraxia/echolalia: relevance to Gilles de la Tourette syndrome. Mov Disord 2012;27(10):1222–1229.
101. Ganos C, Erro R, Cavanna AE, Bhatia KP. Functional tics and echophenomena. Parkinsonism Relat Disord 2014;20(12): 1440–1441.
102. Lees AJ, Robertson M, Trimble MR, Murray NM. A clinical study of Gilles de la Tourette syndrome in the United Kingdom. J Neurol Neurosurg Psychiatry 1984;47(1):1–8.
103. Baruffi MR, de Souza DH, Bicudo da Silva RA, Ramos ES, Moretti-Ferreira D. Autism spectrum disorder in a girl with a de novo x;19 balanced translocation. Case Rep Genet 2012;2012: 578018.
104. Saad AG de F, Goldfeld M. Echolalia in the language development of autistic individuals: a bibliographical review. Pro Fono 2009; 21(3):255–260.
105. van Santen JPH, Sproat RW, Hill AP. Quantifying repetitive speech in autism spectrum disorders and language impairment. Autism Res 2013;6(5):372–383.
106. Klykylo WM, Feldis D, O'Grady D, Ross DL, Halloran C. Clinical effects of fenfluramine in ten autistic subjects. J Autism Dev Disord 1985;15(4):417–423.
107. Chung BI. Brief report: treatment of echolalia in a girl with Rubinstein-Taybi syndrome: functional assessment of minimizing chances to provoke echolalia. J Autism Dev Disord 1998;28(6): 573–578.
108. Levy Y, Gottesman R, Borochowitz Z, Frydman M, Sagi M. Language in boys with fragile X syndrome. J Child Lang 2006;33(1): 125–144.
109. Largo RH, Schinzel A. Developmental and behavioural disturbances in 13 boys with fragile X syndrome. Eur J Pediatr 1985; 143(4):269–275.
110. Rossi NF, Giacheti CM. Association between speech-language, general cognitive functioning and behavior problems in individuals with Williams syndrome. J Intellect Disabil Res 2017;61(7): 707–718.
111. Fekete R. Renal failure in dementia with lewy bodies presenting as catatonia. Case Rep Neurol 2013;5(1):10–13.
112. Miki T, Yokota O, Takenoshita S, et al. Frontotemporal lobar degeneration due to P301L tau mutation showing apathy and severe frontal atrophy but lacking other behavioral changes: A case report and literature review. Neuropathology 2018;38(3):268–280.
113. Valverde AH, Jimenez-Escrig A, Gobernado J, Baron M. A short neuropsychologic and cognitive evaluation of frontotemporal dementia. Clin Neurol Neurosurg 2009;111(3):251–355.
114. Bathgate D, Snowden JS, Varma A, Blackshaw A, Neary D. Behaviour in frontotemporal dementia, Alzheimer's disease and vascular dementia. Acta Neurol Scand 2001;103(6):367–378.
115. Paucar M, Beniaminov S, Paslawski W, Svenningsson P. PSP-CBS with Dopamine Deficiency in a Female with a FMR1 Premutation. Cerebellum 2016;15(5):636–640.
116. Mimura M, Oda T, Tsuchiya K, et al. Corticobasal degeneration presenting with nonfluent primary progressive aphasia: a clinicopathological study. J Neurol Sci 2001;183(1):19–26.
117. Lanska DJ, Currier RD, Cohen M, et al. Familial progressive subcortical gliosis. Neurology 1994;44(9):1633–1643.
118. Saldert C, Hartelius L. Echolalia or functional repetition in conversation–a case study of an individual with Huntington's disease. Disabil Rehabil 2011;33(3):253–260.
119. McPherson SE, Kuratani JD, Cummings JL, Shih J, Mischel PS, Vinters HV. Creutzfeldt-Jakob disease with mixed transcortical aphasia: insights into echolalia. Behav Neurol 1994;7(3):197–203.
120. Lappas AS, Jan F. Unusual psychotic presentation after discontinuation of treatment in a patient with Wilson's disease: a case report.

Clin Schizophr Relat Psychoses 2016; https://doi.org/10.3371/CSRP.LAJA.112316.

121. Dahlqvist G, Guillen-Anaya MA, Vincent MF, Thissen JP, Hainaut P. D-lactic acidosis: an unusual cause of encephalopathy in a patient with short bowel syndrome. Acta Clin Belg 2013; 68(3):229–231.

122. Koff JM, Matsumoto CS, Holtzmuller KC. Echolalia in a liver transplant recipient. Transplantation 2004;78(3):486–487.

123. Seetharam P, Akerman RR. Postoperative echolalia and catatonia responsive to gamma aminobutyric acid receptor agonists in a liver transplant patient. Anesth Analg 2006;103(3):785–786.

124. Hadano K, Nakamura H, Hamanaka T. Effortful echolalia. Cortex 1998;34(1):67–82.

125. Meremikwu MM, Asindi AA, Ezedinachi E. The pattern of neurological sequelae of childhood cerebral malaria among survivors in Calabar, Nigeria. Cent Afr J Med 1997;43(8):231–234.

126. Goldberg EM, Titulaer M, de Blank PM, Sievert A, Ryan N. Anti-N-methyl-D-aspartate receptor-mediated encephalitis in infants and toddlers: case report and review of the literature. Pediatr Neurol 2014;50(2):181–184.

127. Zapor M, Murphy FT, Enzenauer R. Echolalia as a novel manifestation of neuropsychiatric systemic lupus erythematosus. South Med J 2001;94(1):70–72.

128. Karthik MS, Nandhini K, Subashini V, Balakrishnan R. Hashimoto's Encephalopathy Presenting with Unusual Behavioural Disturbances in an Adolescent Girl. Case Rep Med 2017;2017: 3494310.

129. Breit S, Keseru B, Nyffeler T, Sturzenegger M, Krestel H. Posterior fossa syndrome with a large inflammatory ponto-mesencephalic lesion. Brain Cogn 2017;111:107–111.

130. Arya S, Sukhija G, Singh H. Acute Psychosis after Recent Isoniazid Initiation. J Clin Diagn Res 2015;9(6):VD01–2.

131. Chung AM, Reed MD. Intentional topiramate ingestion in an adolescent female. Ann Pharmacother 2004;38(9):1439–1442.

132. Thomas RJ, Reagan DR. Association of a Tourette-like syndrome with ofloxacin. Ann Pharmacother 1996;30(2):138–141.

133. Hofer KE, Degrandi C, Muller DM, et al. Acute toxicity associated with the recreational use of the novel dissociative psychoactive substance methoxphenidine. Clin Toxicol (Phila) 2014;52(10): 1288–1291.

134. Anbarasan D, Campion P, Howard J. Drug-induced leukoencephalopathy presenting as catatonia. Gen Hosp Psychiatry 2011;33(1):85.e1–e3.

135. Itokawa M, Iwata K, Takahashi M, et al. Acute confusional state after designer tryptamine abuse. Psychiatry Clin Neurosci 2007; 61(2):196–199.

136. Tong TG, Benowitz NL, Becker CE, Forni PJ, Boerner U. Phencyclidine poisoning. JAMA 1975;234(5):512–513.

137. Bhati MT, Datto CJ, O'Reardon JP. Clinical manifestations, diagnosis, and empirical treatments for catatonia. Psychiatry (Edgmont) 2007;4(3):46–52.

138. Saint-Hilaire M-H, Saint-Hilaire J-M. Jumping Frenchmen of Maine: Jumping Frenchmen of Maine. Mov Disord 2001;16(3): 530–530.

139. McFarling DA. The "Ragin" Cajuns" of Louisiana: The 'Ragin" Cajuns" of Lousiana.'" Mov Disord 2001;16(3):531–532.

140. Freeman RD, Zinner SH, Muller-Vahl KR, et al. Coprophenomena in Tourette syndrome. Dev Med Child Neurol 2009;51(3): 218–227.

141. de Araujo Lima TF, da Silva Behrens NSC, Lopes E, et al. Kleine-Levin Syndrome: A case report. Sleep Sci 2014;7(2):122–125.

142. Ringman JM, Kwon E, Flores DL, Rotko C, Mendez MF, Lu P. The use of profanity during letter fluency tasks in frontotemporal dementia and Alzheimer disease. Cogn Behav Neurol 2010;23(3): 159–164.

143. Cohen-Mansfield J, Marx MS, Rosenthal AS. A description of agitation in a nursing home. J Gerontol 1989;44(3):M77–M84.

144. Ruiz-Sandoval JL, Garcia-Navarro V, Chiquete E, et al. Choreoacanthocytosis in a Mexican family. Arch Neurol 2007; 64(11):1661–1664.

145. Massot-Tarrus A, Mousavi SR, Dove C, et al. Coprolalia as a manifestation of epileptic seizures. Epilepsy Behav 2016;60:99–106.

146. Edwards MJ, Lang AE, Bhatia KP. Stereotypies: a critical appraisal and suggestion of a clinically useful definition. Mov Disord 2012; 27(2):179–185.

147. Werry JS, Carlielle J, Fitzpatrick J. Rhythmic motor activities (stereotypies) in children under five: etiology and prevalence. J Am Acad Child Psychiatry 1983;22(4):329–336.

148. Temudo T, Oliveira P, Santos M, et al. Stereotypies in Rett syndrome: analysis of 83 patients with and without detected MECP2 mutations. Neurology 2007;68(15):1183–1187.

149. Wong SE, Terranova MD, Bowen L, Zarate R, Massel HK, Liberman RP. Providing independent recreational activities to reduce stereotypic vocalizations in chronic schizophrenics. J Appl Behav Anal 1987;20(1):77–81.

150. Sagar F, Malik SU, Soontornprueksa S, et al. Extrapyramidal Symptoms with Administration of Lenalidomide Maintenance Therapy for Multiple Myeloma. Cureus [Internet]. 2018. https://www.cureus.com/articles/11866-extrapyramidal-symptoms-with-administration-of-lenalidomide-maintenance-therapy-for-multiple-myeloma.

151. Hardie RJ, Pullon HW, Harding AE, et al. Neuroacanthocytosis. A clinical, haematological and pathological study of 19 cases. Brain 1991;114(Pt 1A):13–49.

152. de Teixeira AL, Cardoso F, Maia DP, et al. Frequency and significance of vocalizations in Sydenham's chorea. Parkinsonism Relat Disord 2009;15(1):62–63.

153. Yusupov A, Galvin JE. Vocalization in dementia: a case report and review of the literature. Case Rep Neurol 2014;6(1):126–133.

154. Nagaratnam N, Patel I, Whelan C. Screaming, shrieking and muttering: the noise-makers amongst dementia patients. Arch Gerontol Geriatr 2003;36(3):247–258.

155. von Gunten A, Favre M, Gurtner C, Abderhalden C. Vocally disruptive behavior (VDB) in the institutionalized elderly: A naturalistic multiple case report. Arch Gerontol Geriatr 2011;52(3): e110–e116.

156. Lim S-Y, Tan AH, Lim JL, Ahmad-Annuar A. Purposeless Groaning in Parkinson's Disease. J Mov Disord 2018;11(2):87–88.

157. Low SC, Tan AH, Lim S-Y. Teaching Video NeuroImages: Purposeless groaning in progressive supranuclear palsy. Vol. 88. United States; 2017.

158. Stamelou M, Rubio-Agusti I, Quinn N, Bhatia K. Characteristic constant groaning in late stage progressive supranuclear palsy: a case report. Parkinsonism Relat Disord 2011;17(7):575–576.

159. Mainka T, Hidding U, Buhmann C, Haggard P, Ganos C. Voluntary Inhibition of Involuntary Groaning in Progressive Supranuclear Palsy: Voluntary Inhibition of Involuntary Groaning. Mov Disord Clin Pract 2018;5(3):325–326.

160. Cavanna AE, Ali F, Leckman JF, Robertson MM. Pathological laughter in Gilles de la Tourette syndrome: an unusual phonic tic. Mov Disord 2010;25(13):2233–2239.

161. Neubert G, von Au K, Drossel K, et al. Angelman syndrome and severe infections in a patient with de novo 15q11.2-q13.1 deletion and maternally inherited 2q21.3 microdeletion. Gene 2013;512(2): 453–455.

162. Kortüm F, Das S, Flindt M, et al. The core FOXG1 syndrome phenotype consists of postnatal microcephaly, severe mental retardation, absent language, dyskinesia, and corpus callosum hypogenesis. J Med Genet 2011;48(6):396–406.

163. Olney NT, Goodkind MS, Lomen-Hoerth C, et al. Behaviour, physiology and experience of pathological laughing and crying in amyotrophic lateral sclerosis. Brain 2011;134(Pt 12):3458–3469.

164. Pressman PS, Simpson M, Gola K, et al. Observing conversational laughter in frontotemporal dementia. J Neurol Neurosurg Psychiatry 2017;88(5):418–424.

165. Starkstein SE, Migliorelli R, Tesón A, et al. Prevalence and clinical correlates of pathological affective display in Alzheimer's disease. J Neurol Neurosurg Psychiatry 1995;59(1):55–60.

166. Rohrer JD, Warren JD, Rossor MN. Abnormal laughter-like vocalisations replacing speech in primary progressive aphasia. J Neurol Sci 2009;284(1–2):120–123.

167. Parvizi J, Joseph J, Press DZ, Schmahmann JD. Pathological laughter and crying in patients with multiple system atrophy-cerebellar type. Mov Disord 2007;22(6):798–803.

168. Iwasaki Y, Mori K, Ito M, et al. An autopsy case of Creutzfeldt-Jakob disease with a prion protein gene codon 180 mutation presenting with pathological laughing and an exaggerated startle reaction. Neuropathology 2017;37(6):575–581.

169. Lee HM, Pyo SJ, Kwon KY, Koh S-B. Predominant pathologic laughing and crying in a SCA17 patient. Parkinsonism Relat Disord 2015;21(5):547–548.

170. Wang G, Teng F, Chen Y, et al. Clinical Features and Related Factors of Poststroke Pathological Laughing and Crying: A Case-Control Study. J Stroke Cerebrovasc Dis 2016;25(3):556–564.

171. Dulamea AO, Matei C, Mindruta I, Ionescu V. Pathological laughter as prodromal manifestation of transient ischemic attacks–case report and brief review. BMC Neurol 2015;15:196.

172. Arif H, Mohr JP, Elkind MSV. Stimulus-induced pathologic laughter due to basilar artery dissection. Neurology 2005;64(12): 2154–2155.

173. Dabby R, Watemberg N, Lampl Y, Eilam A, Rapaport A, Sadeh M. Pathological laughter as a symptom of midbrain infarction. Behav Neurol 2004;15(3–4):73–76.

174. Garg RK, Misra S, Verma R. Pathological laughter as heralding manifestation of left middle cerebral artery territory infarct: case report and review of literature. Neurol India 2000;48(4):388–390.

175. Tei H, Sakamoto Y. Pontine infarction due to basilar artery stenosis presenting as pathological laughter. Neuroradiology 1997; 39(3):190–191.

176. Wali GM. "Fou rire prodromique" heralding a brainstem stroke. J Neurol Neurosurg Psychiatry 1993;56(2):209–210.

177. Özel G, Maltête D, Lefaucheur R. Pathological Laughter as a Symptom of Middle Cerebral Artery Stroke. J Emerg Med 2018; 55(5):707–709.

178. Garcia-Baran D, Johnson TM, Wagner J, Shen J, Geers M. Therapeutic Approach of a High Functioning Individual With Traumatic Brain Injury and Subsequent Emotional Volatility With Features of Pathological Laughter and Crying With Dextromethorphan/Quinidine. Medicine (Baltimore) 2016;95(12):e2886.

179. Chahine LM, Chemali Z. Du rire aux larmes: pathological laughing and crying in patients with traumatic brain injury and treatment with lamotrigine. Epilepsy Behav 2006;8(3):610–615.

180. Zeilig G, Drubach DA, Katz-Zeilig M, Karatinos J. Pathological laughter and crying in patients with closed traumatic brain injury. Brain Inj 1996;10(8):591–597.

181. Li Z, Luo S, Ou J, Huang R, Wang Y. Persistent pseudobulbar affect secondary to acute disseminated encephalomyelitis. Socioaffect Neurosci Psychol 2015;5:26210.

182. Chaudhry N, Puri V, Patidar Y, Khwaja GA. Pathological laughter associated with paroxysmal kinesigenic dyskinesia: A rare presentation of acute disseminated encephalomyelitis. Epilepsy Behav Case Rep 2013;1:14–19.

183. de Seze J, Zephir H, Hautecoeur P, Mackowiak A, Cabaret M, Vermersch P. Pathologic laughing and intractable hiccups can occur early in multiple sclerosis. Neurology. 2006;67(9): 1684–1686.

184. Jacob PC, Chand RP. Pathological laughter following intravenous sodium valproate. Can J Neurol Sci 1998;25(3):252–253.

185. Tran TPY, Truong VT, Wilk M, et al. Different localizations underlying cortical gelastic epilepsy: case series and review of literature. Epilepsy Behav 2014;35:34–41.

186. Parvizi J, Le S, Foster BL, et al. Gelastic epilepsy and hypothalamic hamartomas: neuroanatomical analysis of brain lesions in 100 patients. Brain 2011;134(Pt 10):2960–2968.

187. Hogan MB, Wilson NW. Tourette's syndrome mimicking asthma. J Asthma 1999;36(3):253–256.

188. Tan H, Büyükavci M, Arik A. Tourette's syndrome manifests as chronic persistent cough. Yonsei Med J 2004;45(1):145–149.

189. Kempster PA, Lees AJ, Crichton P, Frankel JP, Shorvon P. Off-period belching due to a reversible disturbance of oesophageal motility in Parkinson's disease and its treatment with apomorphine. Mov Disord 1989;4(1):47–52.

190. Mandalà M, Rufa A, Cerase A, et al. Lateral Medullary Ischemia Presenting with Persistent Hiccups and Vertigo. International J Neurosci 2010;120(3):226–230.

191. Delèvaux I, André M, Marroun I, Lamaison D, Piette JC, Aumaître O. Intractable hiccup as the initial presenting feature of systemic lupus erythematosus. Lupus 2005;14(5):406–408.

192. Sethi NK, Torgovnick J, Sethi PK, Arsura E. Nonconvulsive status epilepticus presenting with throat clearing as part of clinical seizure semiology. Clin EEG Neurosci 2010;41(1):50–52.

193. Mestre TA, Bentes C, Pimentel J. Ictal eructation: a case report. Epileptic Disord 2008;10(2):170–172.

194. Gavvala JR, Gerard EE, Macken M, Schuele SU. Seizure ending signs in patients with dyscognitive focal seizures. Epileptic Disord 2015;17(3):255–262.

195. Scheid R, Teich N, Schroeter ML. Aerophagia and belching after herpes simplex encephalitis. Cogn Behav Neurol 2008;21(1): 52–54.

196. Popkirov S, Grönheit W, Wellmer J. Paroxysmal belching: Epileptic or nonepileptic? Epilepsy Behav Case Rep 2016;5:11–12.

197. Brown BJ, Kim S, Saunders H, et al. A Neural Basis for Contagious Yawning. Curr Biol 2017;27(17):2713–2717.e2.

198. Passie T, Hartmann U, Schneider U, Emrich HM. On the function of groaning and hyperventilation during sexual intercourse: intensification of sexual experience by altering brain metabolism through hypocapnia. Med Hypotheses 2003;60(5):660–663.

199. Brown P. The startle syndrome. Mov Disord 2002;17(Suppl 2): S79–S82.

200. Elzawahry H, Do CS, Lin K, Benbadis SR. The diagnostic utility of the ictal cry. Epilepsy Behav 2010;18(3):306–307.

201. Panunzi S, Cardona F, De Liso P, Brinciotti M, Cavanna AE. Ictal coprolalia in a patient with temporal lobe epilepsy. J Neuropsychiatry Clin Neurosci 2013;25(4):E48–E49.

202. Daniel C, Perry MS. Ictal Coprolalia: A Case Report and Review of Ictal Speech as a Localizing Feature in Epilepsy. Pediatr Neurol 2016;57:88–90.

203. Caplan R, Comair Y, Shewmon DA, Jackson L, Chugani HT, Peacock WJ. Intractable seizures, compulsions, and coprolalia: a pediatric case study. J Neuropsychiatry Clin Neurosci 1992;4(3): 315–319.

204. Linetsky E, Planer D, Ben-Hur T. Echolalia-palilalia as the sole manifestation of nonconvulsive status epilepticus. Neurology 2000; 55(5):733–734.

205. Patra S, Elisevich K, Spanaki-Varelas M, Gaddam S, Smith BJ. Ictal barking as a manifestation of temporal lobe epilepsy. Epilepsy Behav 2011;22(2):407–409.

206. Kurian M, Heritier Barras A-C, Korff CM. A child with ictal vocalizations and generalized epilepsy. Epileptic Disord 2015;17(1): 67–70; quiz 71.

207. Doherty MJ, Wilensky AJ, Holmes MD, Lewis DH, Rae J, Cohn GH. Singing seizures. Neurology. 2002;59(9):1435–1438.

208. Kuscu DY, Kayrak N, Karasu A, Gul G, Kirbas D. Ictal singing due to left mesial temporal sclerosis. Epileptic Disord 2008;10(2): 173–176.

209. Bartolomei F, McGonigal A, Guye M, Guedj E, Chauvel P. Clinical and anatomic characteristics of humming and singing in partial seizures. Neurology 2007;69(5):490–492.

210. Robin IG. Snoring. SAGE Publications; 1948.

211. Iriarte J, Campo A, Alegre M, Fernández S, Urrestarazu E. Catathrenia: respiratory disorder or parasomnia? Sleep Med 2015; 16(7):827–830.

212. Giannini G, Calandra-Buonaura G, Mastrolilli F, Righini M, Bacchi-Reggiani ML, Cecere A, et al. Early stridor onset and stridor treatment predict survival in 136 patients with MSA. Neurology 2016;87(13):1375–1383.

213. Kim KJ, Kim J-M, Bae YJ, Yoon I-Y, Song YS, Kim SE. Occurrence of Stridor During Sleep in a Patient With Spinocerebellar Ataxia Type 17. J Clin Sleep Med 2019;15(1):153–155.

214. Heidbreder A, Philipp K. Anti-IgLON 5 Disease. Curr Treat Options Neurol 2018;20(8):29.

215. Trajanovic NN, Voloh I, Shapiro CM, Sandor P. REM sleep behaviour disorder in a child with Tourette's syndrome. Can J Neurol Sci 2004;31(4):572–575.

216. Thirumalai SS, Shubin RA, Robinson R. Rapid eye movement sleep behavior disorder in children with autism. J Child Neurol 2002; 17(3):173–178.

217. Arnaldi D, Antelmi E, St. Louis EK, Postuma RB, Arnulf I. Idiopathic REM sleep behavior disorder and neurodegenerative risk: To tell or not to tell to the patient? How to minimize the risk? Sleep Med Rev 2017;36:82–95.

218. Barone DA, Henchcliffe C. Rapid eye movement sleep behavior disorder and the link to alpha-synucleinopathies. Clin Neurophysiol 2018;129(8):1551–1564.

219. Munhoz RP, Teive HA. REM sleep behaviour disorder: how useful is it for the differential diagnosis of parkinsonism? Clin Neurol Neurosurg 2014;127:71–74.

220. Olson EJ, Boeve BF, Silber MH. Rapid eye movement sleep behaviour disorder: demographic, clinical and laboratory findings in 93 cases. Brain 2000;123(Pt 2):331–339.

221. Cock VC, Lannuzel A, Verhaeghe S, et al. REM sleep behavior disorder in patients with guadeloupean parkinsonism, a tauopathy. Sleep 2007;30(8):1026–1032.

222. Lo Coco D, Cupidi C, Mattaliano A, Baiamonte V, Realmuto S, Cannizzaro E. REM sleep behavior disorder in a patient with frontotemporal dementia. Neurol Sci 2012;33(2):371–373.

223. Lo Coco D, Puligheddu M, Mattaliano P, et al. REM sleep behavior disorder and periodic leg movements during sleep in ALS. Acta Neurol Scand 2017;135(2):219–224.

224. Lo Coco D, Caruso G, Mattaliano A. REM sleep behavior disorder in patients with DJ-1 mutations and parkinsonism-dementia-ALS complex. Mov Disord 2009;24(10):1555–1556.

225. Chi N-F, Shiao G-M, Ku H-L, Soong B-W. Sleep disruption in spinocerebellar ataxia type 3: a genetic and polysomnographic study. J Chin Med Assoc 2013;76(1):25–30.

226. Friedman JH. Presumed rapid eye movement behavior disorder in Machado-Joseph disease (spinocerebellar ataxia type 3). Mov Disord 2002;17(6):1350–1353.

227. Kohyama J, Shimohira M, Kondo S, et al. Motor disturbance during REM sleep in group A xeroderma pigmentosum. Acta Neurol Scand 1995;92(1):91–95.

228. Arnulf I, Nielsen J, Lohmann E, Schiefer J, Schieffer J, Wild E, et al. Rapid eye movement sleep disturbances in Huntington disease. Arch Neurol 2008;65(4):482–488.

229. Culebras A, Moore JT. Magnetic resonance findings in REM sleep behavior disorder. Neurology 1989;39(11):1519–1523.

230. Kimura K, Tachibana N, Kohyama J, Otsuka Y, Fukazawa S, Waki R. A discrete pontine ischemic lesion could cause REM sleep behavior disorder. Neurology 2000;55(6):894–895.

231. Xi Z, Luning W. REM sleep behavior disorder in a patient with pontine stroke. Sleep Med 2009;10(1):143–146.

232. Zambelis T, Paparrigopoulos T, Soldatos CR. REM sleep behaviour disorder associated with a neurinoma of the left pontocerebellar angle. J Neurol Neurosurg Psychiatry 2002;72(6): 821–822.

233. Plazzi G, Montagna P. Remitting REM sleep behavior disorder as the initial sign of multiple sclerosis. Sleep Med 2002;3(5):437–439.

234. Cochen V, Arnulf I, Demeret S, et al. Vivid dreams, hallucinations, psychosis and REM sleep in Guillain-Barré syndrome. Brain 2005; 128(Pt 11):2535–2545.

235. Compta Y, Iranzo A, Santamaría J, Casamitjana R, Graus F. REM sleep behavior disorder and narcoleptic features in anti-Ma2-associated encephalitis. Sleep 2007;30(6):767–769.

236. Schenck CH, Mahowald MW. Motor dyscontrol in narcolepsy: rapid-eye-movement (REM) sleep without atonia and REM sleep behavior disorder. Ann Neurol 1992;32(1):3–10.

237. Manni R, Terzaghi M. REM behavior disorder associated with epileptic seizures. Neurology 2005;64(5):883–884.

238. Husain AM, Miller PP, Carwile ST. Rem sleep behavior disorder: potential relationship to post-traumatic stress disorder. J Clin Neurophysiol 2001;18(2):148–157.

239. Vela-Bueno A, Soldatos CR. Episodic sleep disorders (parasomnias). Semin Neurol 1987;7(3):269–276.

240. Menghi V, Bisulli F, Tinuper P, Nobili L. Sleep-related hypermotor epilepsy: prevalence, impact and management strategies. Nat Sci Sleep 2018;10:317–326.

241. Jürgens U. Neural pathways underlying vocal control. Neurosci Biobehav Rev 2002;26(2):235–258.

242. Jürgens U. The Neural Control of Vocalization in Mammals: A Review. J Voice 2009;23(1):1–10.

243. McCairn KW, Bronfeld M, Belelovsky K, Bar-Gad I. The neurophysiological correlates of motor tics following focal striatal disinhibition. Brain 2009;132(Pt 8):2125–2138.

244. Bronfeld M, Yael D, Belelovsky K, Bar-Gad I. Motor tics evoked by striatal disinhibition in the rat. Front Syst Neurosci 2013;7:50.

245. Kalanithi PSA, Zheng W, Kataoka Y, et al. Altered parvalbumin-positive neuron distribution in basal ganglia of individuals with Tourette syndrome. Proc Natl Acad Sci U S A 2005;102(37): 13307–13312.

246. Kataoka Y, Kalanithi PSA, Grantz H, et al. Decreased number of parvalbumin and cholinergic interneurons in the striatum of individuals with Tourette syndrome. J Comp Neurol 2010;518(3):277–291.

247. McCairn KW, Nagai Y, Hori Y, et al. A Primary Role for Nucleus Accumbens and Related Limbic Network in Vocal Tics. Neuron 2016;89(2):300–307.

248. Maia TV, Conceição VA. The Roles of Phasic and Tonic Dopamine in Tic Learning and Expression. Biol Psychiatry. 2017;82(6):401–412.

249. Martino D, Hedderly T. Tics and stereotypies: A comparative clinical review. Parkinsonism & Related Disorders [Internet]. 2019. https://linkinghub.elsevier.com/retrieve/pii/S1353802019300367. Accessed March 6, 2019

250. Kates WR, Lanham DC, Singer HS. Frontal white matter reductions in healthy males with complex stereotypies. Ped Neurol 2005; 32(2):109–112.

251. Péter Z, Oliphant ME, Fernandez TV. Motor Stereotypies: A Pathophysiological Review. Frontiers in Neuroscience [Internet]. 2017. http://journal.frontiersin.org/article/10.3389/fnins.2017.00171/full. Accessed March 9, 2019.

252. Lutz CK. Stereotypic Behavior in Nonhuman Primates as a Model for the Human Condition. ILAR J 2014;55(2):284–296.

253. Hutt C, Hutt SJ. Effects of environmental complexity on stereotyped behaviours of children. Animal Behav 1965;13(1):1–4.

254. Kaji R, Bhatia K, Graybiel AM. Pathogenesis of dystonia: is it of cerebellar or basal ganglia origin? J Neurol Neurosurg Psychiatry 2018;89(5):488–492.

255. Albin RL. The pathophysiology of chorea/ballism and Parkinsonism. Parkinsonism Relat Disord 1995;1(1):3–11.

256. Madeo G, Pisani A. Pathophysiology of dystonia. In: Handbook of Behavioral Neuroscience [Internet]. Elsevier; 2016:929–950. https://linkinghub.elsevier.com/retrieve/pii/B9780128022061000477.

257. Factor SA, Lang AE, Weiner WJ, editors. Drug Induced Movement Disorders [Internet]. Malden, Massachusetts: Blackwell Publishing Inc.; 2005. http://doi.wiley.com/10.1002/9780470753217. Accessed July 16, 2019.

258. Teo JT, Edwards MJ, Bhatia K. Tardive dyskinesia is caused by maladaptive synaptic plasticity: a hypothesis. Mov Disord 2012; 27(10):1205–1215.

259. Gay F, Engelhardt M, Terpos E, et al. From transplant to novel cellular therapies in multiple myeloma: European Myeloma Network guidelines and future perspectives. Haematologica 2018;103(2):197–211.

260. Lanctôt KL, Herrmann N, Mazzotta P. Role of serotonin in the behavioral and psychological symptoms of dementia. J Neuropsychiatry Clin Neurosci 2001;13(1):5–21.

261. Téllez-Zenteno JF, Serrano-Almeida C, Moien-Afshari F. Gelastic seizures associated with hypothalamic hamartomas. An update in the clinical presentation, diagnosis and treatment. Neuropsychiatr Dis Treat 2008;4(6):1021–1031.

262. Kovac S, Diehl B, Wehner T, et al. Gelastic seizures: Incidence, clinical and EEG features in adult patients undergoing video-EEG telemetry. Epilepsia 2015;56(1):e1–e5.

263. Mohamed IS, Otsubo H, Shroff M, Donner E, Drake J, Snead OC. Magnetoencephalography and diffusion tensor imaging in gelastic seizures secondary to a cingulate gyrus lesion. Clin Neurol Neurosurg 2007;109(2):182–187.

264. McConachie NS, King MD. Gelastic seizures in a child with focal cortical dysplasia of the cingulate gyrus. Neuroradiology. 1997; 39(1):44–45.

265. García A, Gutiérrez MA, Barrasa J, Herranz JL. Cryptogenic gelastic epilepsy of frontal lobe origin: a paediatric case report. Seizure 2000;9(4):297–300.

266. Sartori E, Biraben A, Taussig D, Bernard AM, Scarabin JM. Gelastic seizures: video-EEG and scintigraphic analysis of a case with a frontal focus; review of the literature and pathophysiological hypotheses. Epileptic Disord 1999;1(4):221–228.

267. Shin H-Y, Hong SB, Joo EY, et al. Gelastic seizures involving the right parietal lobe. Epileptic Disord 2006;8(3):209–212.

268. Dericioglu N, Cataltepe O, Tezel GG, Saygi S. Gelastic seizures due to right temporal cortical dysplasia. Epileptic Disord 2005; 7(2):137–141.

269. Voon V, Cavanna AE, Coburn K, Sampson S, Reeve A, LaFrance WC, et al. Functional Neuroanatomy and Neurophysiology of Functional Neurological Disorders (Conversion Disorder). J Neuropsychiatry Clin Neurosci 2016;28(3):168–190.

270. Edwards MJ, Adams RA, Brown H, Pareés I, Friston KJ. A Bayesian account of "hysteria." Brain 2012;135(Pt 11):3495–3512.

271. Baizabal-Carvallo JF, Hallett M, Jankovic J. Pathogenesis and pathophysiology of functional (psychogenic) movement disorders. Neurobiol Dis 2019;127:32–44.

272. Horvath RA, Fogarasi A, Schulz R, et al. Ictal vocalizations occur more often in temporal lobe epilepsy with dominant (left-sided) epileptogenic zone. Epilepsia 2009;50(6):1542–1546.

273. Iranzo A. The REM sleep circuit and how its impairment leads to REM sleep behavior disorder. Cell Tissue Res 2018;373(1):245–266.

274. García-Lorenzo D, Longo-Dos Santos C, Ewenczyk C, et al. The coeruleus/subcoeruleus complex in rapid eye movement sleep behaviour disorders in Parkinson's disease. Brain 2013;136(Pt 7):2120–2129.

275. Gagnon J-F, Postuma RB, Mazza S, Doyon J, Montplaisir J. Rapid-eye-movement sleep behaviour disorder and neurodegenerative diseases. Lancet Neurol 2006;5(5):424–432.

276. Fründt O, Woods D, Ganos C. Behavioral therapy for Tourette syndrome and chronic tic disorders. Neurol Clin Pract 2017;7(2): 148–156.

277. Jankovic J, Glaze DG, Frost JD. Effect of tetrabenazine on tics and sleep of Gilles de la Tourette's syndrome. Neurology 1984;34(5):688–692.

278. Jankovic J, Jimenez-Shahed J, Budman C, et al. Deutetrabenazine in Tics Associated with Tourette Syndrome. Tremor Other Hyperkinet Mov (N Y). 2016;6:422.

279. Müller-Vahl KR. Treatment of Tourette syndrome with cannabinoids. Behav Neurol 2013;27(1):119–124.

280. Baldermann JC, Schüller T, Huys D, et al. Deep Brain Stimulation for Tourette-Syndrome: A Systematic Review and Meta-Analysis. Brain Stimul 2016;9(2):296–304.

281. Pandey S, Srivanitchapoom P, Kirubakaran R, Berman BD. Botulinum toxin for motor and phonic tics in Tourette's syndrome. Cochrane Database Syst Rev 2018;1:CD012285.

282. Ganos C, Edwards MJ, Bhatia KP. Posttraumatic functional movement disorders. Handb Clin Neurol 2016;139:499–507.

283. Serra-Mestres J, Shapleske J, Tym E. Treatment of palilalia with trazodone. Am J Psychiatry 1996;153(4):580–581.

284. Berthier ML, Torres-Prioris MJ, Lopez-Barroso D. Thinking on Treating Echolalia in Aphasia: Recommendations and Caveats for Future Research Directions. Front Hum Neurosci 2017;11:164.

285. McMinn B, Draper B. Vocally disruptive behaviour in dementia: development of an evidence based practice guideline. Aging Ment Health 2005;9(1):16–24.

286. Ramadan FH, Naughton BJ, Bassanelli AG. Treatment of verbal agitation with a selective serotonin reuptake inhibitor. J Geriatr Psychiatry Neurol 2000;13(2):56–59.

287. Pollock BG, Mulsant BH, Sweet R, et al. An open pilot study of citalopram for behavioral disturbances of dementia. Plasma levels and real-time observations. Am J Geriatr Psychiatry 1997;5(1):70–78.

288. Hottin P. [Pharmacotherapy to control agitation in patients with cognitive deficits]. Can J Psychiatry 1990;35(3):270–272.

289. Greenwald BS, Marin DB, Silverman SM. Serotoninergic treatment of screaming and banging in dementia. Lancet 1986;2(8521–22): 1464–1465.

290. Friedman R, Gryfe CI, Tal DT, Freedman M. The noisy elderly patient: prevalence, assessment, and response to the antidepressant doxepin. J Geriatr Psychiatry Neurol 1992;5(4):187–191.

291. Müller U, Murai T, Bauer-Wittmund T, von Cramon DY. Paroxetine versus citalopram treatment of pathological crying after brain injury. Brain Inj 1999;13(10):805–811.

292. Gilson I, Busalacchi M. Marijuana for intractable hiccups. Lancet 1998;351(9098):267.

293. Bhatia KP, Erro R, Stamelou M. Case Studies in Movement Disorders: Common and Uncommon Presentations [Internet]. Cambridge, UK: Cambridge University Press; 2017. http://ebooks.cambridge.org/ref/id/CBO9781316145050. Accessed April 9, 2019.

294. Tijssen MAJ, Brown P, Morris HR, Lees A. Late onset startle induced tics. J Neurol Neurosurg Psychiatry 1999;67(6):782–784.

295. Ganos C, Erro R, Cavanna AE, Bhatia KP. Functional tics and echophenomena. Parkinsonism Relat Disord 2014;20(12):1440–1441.

Supporting Data

Additional Supporting Information may be found in the online version of this article at the publisher's web-site.